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THE STUDY OF PCOS IN ENDOCRINOLOGY (THYROID GLAND) AND LIFESTYLE MANAGEMENT

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ABSTRACT

Polycystic ovary syndrome (PCOS) is a complex endocrine and metabolic disorder, typically characterized by anovulation, infertility, obesity, insulin resistance, and polycystic ovaries. Lifestyle or diet, environmental pollutants, genetics, gut dysbiosis, neuroendocrine alterations, and obesity are among the risk factors that predispose females to PCOS. These factors might contribute to upsurging metabolic syndrome by causing hyperinsulinemia, oxidative stress, hyperandrogenism, impaired folliculogenesis and irregular menstrual cycles. Lifestyle modifications and complementary and alternative medicines are preferred first-line therapy in many cases. Medications, including 3-hydroxy-3-methyl-3-glutaryl- coenzyme A (HMG-CoA) reductase inhibitors, thiazolidinediones, sodium-glucose cotransporter-2 inhibitors, dipeptidyl peptidase-4 inhibitors, glucose-like peptide-1 receptor agonists, mucolytic agents, and some supplements have supporting data for being repurposed in PCOS. This article focuses on the thyroid related issues with that of PCOS. This article also deals with the anatomy and physiology of thyroid gland, focusing on the related effects of PCOS on thyroid gland. The PCOS guideline recommends the promotion of healthy lifestyle behaviours in all women with PCOS.

KEYWORDS: Polycystic ovary syndrome, hyperandrogenism, insulin resistance, thyroid gland, lifestyle management.

INTRODUCTION

One of the most prevalent endocrine system conditions affecting women of reproductive age is polycystic ovary syndrome (PCOS), also known as hyperandrogenic anovulation (HA) or Stein–Leventhal syndrome.[1] This chronic and heterogeneous disorder manifests itself as menstrual dysfunction, infertility, hirsutism, acne, and obesity.[2] It describes a condition where at least one ovary has an ovarian volume greater than 10 ml and at least one ovary has an estimated ten small cysts, with diameters ranging from 2 to 9 mm, develop.[3] It is usually only diagnosed when complications develop that significantly reduce a patient's quality of life (e.g. alopecia, acne, and infertility-related problems).[4] According to a systematic screening of women using the National Institutes of Health (NIH) diagnostic standards, 4–10% of reproductive-age women are predicted to have PCOS world wide. The World Health Organization (WHO) estimates that in 2012 PCOS affected 116 million women (3.4%) globally.[5] This high frequency, as well as its link with ovulation and menstruation abnormalities, infertility, hair loss, and metabolic issues, underscores PCOS's significant financial burden.[6] Although PCOS can occur at any age, beginning with menarche, the majority of instances are identified between the ages of 20 and 30.[7] PCOS affects 1.55 million women of reproductive age worldwide, resulting in 0.43 million disability-adjusted life years (DALYs). The age-standardized incidence rate of PCOS in women of reproductive age was 82.44 per 100,000 in 2017, 1.45% higher than in 2007.[8] Recent research reveals that PCOS is a lifelong syndrome that first manifests during pregnancy, although it was traditionally thought to be a disorder that only affected adult women.[9] While the exact cause of this multifactorial disorder is unknown, a combination of inherited and environmental factors is thought to play a primary role. The pathophysiology of PCOS is chiefly concerned with hormonal imbalance, chronic low-grade inflammation, insulin resistance, and hyperandrogenism, which impair folliculogenesis and increase the risk of related commorbidities, such as endometrial cancer and type II diabetes. According to international recommendations, the three main factors used to diagnose PCOS are hyperandrogenism, ovarian morphology, and anovulation.[10] A range of environmental factors, including geography, diet and nutrition, socioeconomic status, and environmental pollutants, are possibly contributing to the development, occurrence, and management of PCOS.[11] In recent years, the link between PCOS and the microbiome has been established, and it is believed to

have contributed to the establishment of the syndrome. Dysbiosis of the gut microbial community, caused by environmental risk factors, might be a potential pathogenic factor in the development and progression of PCOS. Different pathogenic aspects of PCOS are caused by different microbiota, and essential routes linking their involvement in the onset of various clinical manifestations of PCOS bring up new therapy options for the condition.[12] Prebiotics, probiotics, symbiotic, and faecal microbiota transplants (FMTs) help manage the variety of phenotypes associated with PCOS by boosting Eubiosis and reducing the impact of altered microbial profiles. Microbiota-mediated therapies might improve the metabolic, inflammatory, and hormonal characteristics of PCOS women.

This review summarizes the risk factors that may contribute to the development, prevalence, and modulation of PCOS, as well as its possible treatment approaches, including IL-22 and mRNA therapy. Additionally, we discuss the importance of factors contributing to that of thyroid and endocrine gland.

THYROID GLAND

The thyroid, or thyroid gland, is an endocrine gland in vertebrates. In humans, it is a butterfly-shaped gland located in the neck below the Adam's apple. It consists of two connected lobes. The lower two thirds of the lobes are connected by a thin band of tissue called the isthmus. Microscopically, the functional unit of the thyroid gland is the spherical thyroid follicle, lined with follicular cells (thyrocytes), and occasional parafollicular cells that surround alumen containing colloid.

The thyroid gland secretes three hormones: the two thyroid hormones – triiodothyronine (T3) and thyroxine (T4) – and a peptide hormone, calcitonin. The thyroid hormones influence the metabolic rate and protein synthesis and growth and development in children. Calcitonin plays a role in calcium homeostasis.[13]

Secretion of the two thyroid hormones is regulated by thyroid-stimulating hormone (TSH), which is secreted from the anterior pituitary gland. TSH is regulated by thyrotropin-releasing hormone (TRH), which is produced by the hypothalamus.[14]

FEMALE REPRODUCTIVE SYSTEM

The female reproductive system comprises internal and external organs that facilitate menstruation and procreation. This organ system is responsible for producing gametes

(termed eggs or ova), regulating sex hormones, and maintaining fertilized eggs as they develop into mature foetuses ready for delivery.[15] A woman's reproductive years are between menarche (the first menstrual cycle) and menopause (cessation of menses for 12 consecutive months). During this period, cyclical expulsion of ova from the ovary occurs, with the potential for fertilization by male gametes or sperm. This cyclic expulsion of eggs is a normal part of the menstrual cycle. Separately, the internal and external female genitalia facilitate fertilization from the unison of ova and sperm, leading to the process of gestation, also termed pregnancy. Depending on how the conception evolves, typically within 9 months or 3 trimesters, the gestation can produce one or more foetus that, when born, are termed infants.[16]

PATHOPHYSIOLOGY

PCOS is a hyperandrogenic state with oligo-anovulation that any other disorder cannot explain. It is a diagnosis of exclusion. Never the less, it accounts for the majority of hyperandrogenic presentations. Nearly all causes of PCOS are due to functional ovarian hyperandrogenism (FOH). Two-thirds of PCOS presentations have typical functional ovarian hyperandrogenism, characterized by dysregulation of androgen secretion with an over-response of 17-hydroxyprogesterone (17-OHP) to gonadotropin stimulation. The remaining PCOS has an atypical FOH response of 17-OHP, but testosterone elevation can be detected after suppressing adrenal androgen production. About 3% of PCOS patients have a related isolated functional adrenal hyperandrogenism. The remainder of PCOS cases are mild. These lack evidence of steroid secretory abnormalities; most of these patients are obese, which practitioners postulate accounts for their atypical PCOS. Specific testing for the FOH sub population has low clinical utility in the present day.[17]

Functional ovarian hyperandrogenism PCOS presents with the primary features: hyperandrogenism, oligo anovulation, and polycystic ovary morphology. Functional ovarian hyperandrogenism is multifactorial, with a combination of hereditable and environmental factors. Causes for this dysregulation include insulin excess, which is known to sensitize the ovary to luteinizing hormone (LH) by interfering with the process of homologous desensitization to LH in the normal ovulation cycle as well as an intrinsic imbalance among intraovarian regulatory systems. Theca cells in PCOS have overexpression of most steroidogenic enzymes and proteins involved in androgen synthesis, which suggests a prominent abnormality at the level and activity of steroidogenic enzymes, including P450c17,

which has been highly identified. Granulosa cells prematurely luteinize primarily as a result of androgen and insulin excess.[18]

Androgen excess enhances the initial recruitment of primordial follicles into the growth pool. Simultaneously, it initiates premature luteinization and impairs the dominant follicle selection. This results in classical PCOS histopathologic and gross anatomic changes constituting polycystic ovarian morphology (PCOM). Increased LH perpetuates PCOS, but it is not caused by it. LH excess is common and is necessary for the expression of gonadal steroidogenic enzymes and sex hormone secretion but is less likely to be the primary cause of ovarian androgen excess because of LH-induced desensitization of theca cells. About 1-half of patients with functional ovarian hyperandrogenism have an abnormal degree of insulin-resistant hyperinsulinism, which acts on theca cell, increasing steroidogenesis prematurely, luteinizes granulosa cells, and stimulates fat accumulation.[19] Hyperandrogenaemia provokes LH excess, which then acts on theca and luteinized granulosa sustaining cycle.[20]

Ovarian hormonal dysregulation alters the pulsatile gonadotropin-releasing hormone release, potentially leading to a relative increase in LH versus follicle-stimulating hormone (FSH) biosynthesis and secretion. LH stimulates ovarian androgen production, while the relative decrease of FSH prevents adequate stimulation of aromatase activity within the granulosa cells, decreasing androgen conversion to the potent oestrogen oestradiol.[21] This becomes a self-perpetuating, noncyclic hormonal pattern. Elevated serum androgens are converted in the periphery to oestrogens, mostly estrone. As conversion occurs primarily in the stromal cells of adipose tissue, oestrogen production is augmented in obese PCOS patients.[22] This conversion results in chronic feedback at the hypothalamus and pituitary gland, in contrast to the normal fluctuations in feedback observed in the presence of a growing follicle and rapidly changing oestradiol levels. Unopposed oestrogen stimulation of the endometrium may lead to endometrial hyperplasia.[23]

CLINICAL MANIFESTATIONS

The most common signs and symptoms of PCOS include.

- Irregular periods: Abnormal menstruation involves missing periods or not having a period at all. It may also involve heavy bleeding during periods.
- Abnormal hair growth: You may grow excess facial hair or experience heavy hair growth on your arms, chest and abdomen (hirsutism). This affects up to 70% of people with PCOS.

- Acne: PCOS can cause acne, especially on your back, chest and face. This acne may continue past your teenage years and may be difficult to treat.
- Obesity: Between 40% and 80% of people with PCOS have obesity and have trouble maintaining a weight that's healthy for them.
- Darkening of the skin: You may get patches of dark skin, especially in the folds of your neck, armpits, groin (between the legs) and under your breasts. This is known as acanthosis nigricans.
- Cysts: Many people with PCOS have ovaries that appear larger or with many follicles (egg sac cysts) on ultrasound.
- Skin tags: Skin tags are little flaps of extra skin. They're often found in your armpits or on your neck.
- Thinning hair: People with PCOS may lose patches of hair on their head or start to bald.
- Infertility: PCOS is the most common cause of female infertility. Not ovulating regularly or frequently can result in not being able to conceive.

CO-OCCURRENCE OF PCOS AND THYROID DISEASE:

The incidence rate of thyroid diseases is increased in patients with PCOS.[24] In 2019, a study from Denmark reported that the risk of thyroid disease in PCOS patients is 2.5 times higher than that in patients without PCOS[25], therefore, we have thoroughly analysed the comorbidity of several thyroid diseases with PCOS.

Hyperthyroidism

Graves' disease (GD) is the primary clinical cause of hyperthyroidism with global incidences reported between 1.5 and 6.5 per 100,000.[26] It tends to increase in frequency with age. It is predominantly considered to be an organ-specific autoimmune disease[27], characterized by infiltration of the thyroid by T and B lymphocytes that react against thyroid antigens and the production of thyroid autoantibodies. The main thyroid autoantibodies are directed against thyroid-stimulating hormone receptors, which can activate the TSH receptor, leading to excessive production of thyroid hormones. Therefore, patients with this disease typically present with hyperthyroidism and diffuse enlargement of the thyroid gland.[28]

There are few reports that indicate an association between PCOS and GD. Jung et al. was the first to describe a female patient with PCOS and GD in 2011; she presented with decreased menstruation, low body mass index (BMI: 16.4 kg/m^2), mild hirsutism, and thyrotoxicosis.[29] In 2012, six female patients with PCOS and GD were identified in a tertiary care centre in

northern India. They presented with goitre based on clinical and ultrasound examination; furthermore, all women were thin, with an average BMI of 22.73 kg/m^2, and three of the six women had a waist circumference of < 80 cm. Additionally, UMA Sinha et al. reported two patients from India with both PCOS and GD, who showed elevated anti-thyroid peroxidase (TPO) antibody levels. The prevalence of PCOS combined with GD is unclear, because current information is limited to case reports, and extensive epidemiological data is currently lacking. Moreover, the incidence of PCOS combined with GD may differ with race or ethnicity, since the patients referenced in these case reports were all Asian women.[30] The probability of having PCOS combined with dominant or subclinical hyperthyroidism in young female western populations is very low, which may be related to the lower prevalence of hyperthyroidism in the general western population.

Subclinical hypothyroidism

The prevalence of SCH is higher in PCOS patients than that in the general population. Raj et al. conducted a study of Pakistani women 18–30 years old to determine the incidence of SCH in PCOS patients.[31] By comparing 200 PCOS patients with 200 control patients without PCOS, they determined that SCH was more prevalent in PCOS patients (43.5%) than in participants without PCOS (20.5%). They also found that weight gain and BMI of the PCOS patients were significantly higher than those without PCOS.[32]

Since hypothyroidism commonly occurs in PCOS patients, this correlation strongly suggests an increased risk of thyroid disease with PCOS; therefore, it is important to explain its clinical impact. SCH may cause mild metabolic abnormalities. For example, a clinical study of 4,065 PCOS patients 12–40 years of age revealed that more patients with SCH were shown to have obesity, central obesity, and goiter compared with the normal thyroid group.[33] Furthermore, women with SCH are more likely to have abnormal fasting plasma glucose (FPG) levels and insulin resistance index (HOMA-IR) than women without SCH. Additionally, a study of 4,821 participants, comprised of 1,300 PCOS patients with SCH and 3,521 PCOS patients without SCH, found that the HOMA- IR, triglyceride, serum total cholesterol (TC), low density lipoprotein (LDL), fasting blood glucose (FBG), fasting C-peptide (FCP), and prolactin levels were higher, while high-density lipoprotein cholesterol (HDL), luteinizing hormone (LH), and testosterone levels were lower in the SCH patients. Collectively, these results indicate that the incidence of metabolic syndrome is higher in the SCH group, which indicates that SCH may aggravate lipid- and glucose-related metabolic

disorders in PCOS patients.

Thyroiditis

Thyroiditis is a heterogeneous disease of the thyroid gland with various etiologies. Hashimoto's thyroiditis (HT), also known as chronic lymphocytic thyroiditis, is a type of AIT and is a common form of thyroiditis in young women.[34] HT may occur concurrently with clinical hypothyroidism (the most common), normal thyroid function, or hyperthyroidism.

Janssen et al. were the first to confirm that the prevalence of HT in PCOS patients was higher than that in non-PCOS patients through a systematic prospective study with 175 PCOS patients and 168 healthy controls.[35] In 26.9% of PCOS patients and 8.3% of the control group, HT-specific anti-TPO or anti-thyroglobulin (TG) antibody levels were found to be elevated, revealing a threefold increase in the prevalence of HT in PCOS patients relative to controls. Furthermore, thyroid ultrasound examination results revealed that 42.3% of PCOS patients, but only 6.5% of the control group, exhibited a typical HT hypoechoic thyroid ultrasound pattern indicative of mild thyroid damage.

Since HT prevalence is known to be higher in women with PCOS, it is important to understand if the reverse is also true, namely, if the prevalence of PCOS is higher in women with HT than in women without HT. Ganie et al. conducted a prospective case- control study in India on adolescent females 13–18 years old comparing 1,075 HT patients with normal thyroid function and 46 age-matched patients without HT based on negative anti-TPO antibody tests.[36] Their results showed that the prevalence of PCOS was significantly higher in HT patients (46.8%) than in non-HT patients (4.3%). Moreover, the BMI, waist circumference, and systolic blood pressure were all higher in HT patients than in controls.

METHODS

Nine main areas of interest were identified and analysed according to the available evidence: 1) Evaluation of thyroid function for PCOS diagnosis; 2) Epidemiology data on thyroid function/disorders in patients with PCOS, and vice versa; 3) Experimental data supporting the relationship between thyroid function/disorders and PCOS; 4) Effects of thyroid function/disorders on PCOS features, and vice versa; 5) Effect of thyroid alterations on the cardiometabolic risk in women with PCOS; 6) Effect of thyroid abnormalities on reproductive outcomes in women with PCOS; 7) Relationship between thyroid function/abnormalities in patients with PCOS who are undergoing fertility treatment; 8)

Effect of treatments for thyroid diseases on PCOS; and 9) Effect of treatments for PCOS on thyroid function. An extensive literature search for specific keywords was performed for articles published from 1970 to March 2023 using PubMed and Web of Science. Data were reported in a narrative fashion.

DIAGNOSIS

To receive a diagnosis of PCOS, you must meet two of the following criteria.

- irregular ovulation, which is usually indicated by an irregular menstrual cycle or a lack of a cycle
- signs of increased androgen levels or a blood test confirming you have increased levels
- multiple small cysts on the ovaries

To diagnose PCOS, an endocrinologist, a doctor who specializes in hormonal disorders, conducts a physical exam. He or she checks you for increased body and facial hair, thinning scalp hair, acne, and other symptoms of increased androgen levels. The doctor also asks about your medical history, including the regularity of your menstrual cycle.

DIAGNOSTIC TESTS

Blood Tests

Your doctor may check your blood for levels of androgens, including testosterone, which tends to be higher in women with PCOS. He or she may also test your insulin levels, which are usually elevated in women with the condition.

The doctor may also measure levels of the hormones involved in ovulation, such as luteinizing hormone (LH), follicle-stimulating hormone (FSH), and progesterone. These tests can indicate whether you are ovulating.

Your doctor may also test your blood to rule out conditions that mimic PCOS, as well as tumours of the ovaries or adrenal glands. He or she may also check your blood for signs of hypothyroidism, in which the thyroid gland doesn’t produce enough thyroid hormone, leading to fatigue and possibly depression. A blood test can also reveal whether you have high cholesterol and high blood sugar.

Pelvic Ultrasound

Your doctor may recommend a pelvic ultrasound---a test that uses sound waves to create images of the uterus and the ovaries on a computer monitor—to look for ovarian cysts.

However, this test might not show ovarian cysts in women with PCOS who are taking birth control pills.

TREATMENT

Strategies for Incorporating Lifestyle Changes to Manage PCOS Symptoms.

1. Balanced Diet

The cornerstone of PCOS management lies in dietary choices. A well-balanced diet comprising fruits, vegetables, whole grains, lean proteins, and healthy fats delivers vital nutrients while maintaining stable blood sugar levels. Choosing foods with a low glycemic index helps prevent insulin spikes, often associated with PCOS. Additionally, integrating fibre-rich foods like legumes, oats, and flaxseeds can assist in blood sugar regulation and support digestive well-being.

2. Regular Exercise

Regular exercise is a powerful asset in addressing PCOS symptoms. By engaging in consistent physical activity, individuals can not only manage weight effectively but also enhance insulin sensitivity and regulate hormones. Strive for a diverse exercise routine encompassing cardiovascular workouts, strength training, and flexibility exercises to optimize results. Activities such as brisk walking, swimming, cycling, and yoga hold particular promise for women managing PCOS.

3. Stress Management

Persistent stress can disrupt hormone equilibrium, worsening PCOS symptoms. Introducing stress-relieving techniques like mindfulness meditation, deep breathing exercises, or connecting with nature can effectively lower stress levels. Furthermore, prioritizing sufficient sleep and establishing healthy boundaries in both personal and professional spheres can significantly enhance overall well-being.

4. Adequate Hydration

Proper hydration plays a crucial role in supporting metabolic function and hormone equilibrium. Make it a priority to consume ample water throughout the day, and explore options like herbal teas or infused water as alternatives to sugary or caffeinated drinks. Adequate hydration also facilitates the elimination of toxins from the body and promotes healthy skin, offering potential relief for women with PCOS who may contend with acne and

other skin concerns.

5. Restrict Consumption of Caffeine and Alcohol

While an occasional cup of coffee or a glass of wine might not pose significant harm, excessive consumption of caffeine and alcohol can disturb hormone equilibrium and worsen PCOS symptoms. It's advisable to limit your intake or switch to decaffeinated beverages and non-alcoholic alternatives. Herbal teas, fruit-infused sparkling water, and mocktails offer satisfying options without compromising your health.

CONCLUSION

The thyroid diseases associated with PCOS are primarily SCH and AITDs; the co-occurrence of PCOS with hyperthyroidism is relatively rare. This association suggests that the thyroid may affect the clinical manifestations of PCOS by influencing multiple systems including metabolism and immunity. Insulin resistance is a widely recognized cause of PCOS, and thyroid function affects the degree of insulin resistance; specifically, hypothyroidism leads to more serious insulin resistance than hyperthyroidism. Obesity has seriously affected the health of modern people, and patients with hypothyroidism are prone to obesity. The patients with PCOS accompanied by hypothyroidism tend to have high BMI with a corresponding increase in metabolic disease burden. When the thyroid function of patients with SCH or clinical hypothyroidism is restored through thyroid treatment, their metabolic abnormalities improve. In addition, the thyroid also plays an important role in PCOS-related reproductive disorders. In conclusion, the role of the thyroid in PCOS is complex and involves multiple pathways. Thyroid function directly affects the clinical manifestations of PCOS, which increases the heterogeneity of the clinical PCOS phenotype. In fact, clinical reports have shown that some PCOS symptoms have been alleviated or even eliminated by restoring thyroid function. This evidence strongly suggests that the thyroid plays a crucial role in the pathogenesis, development, and progression of PCOS. Therefore, patients with PCOS require rigorous thyroid function detection, monitoring, and correction over time, which will mitigate or perhaps fully prevent the further deterioration of PCOS symptoms.

RESULTS

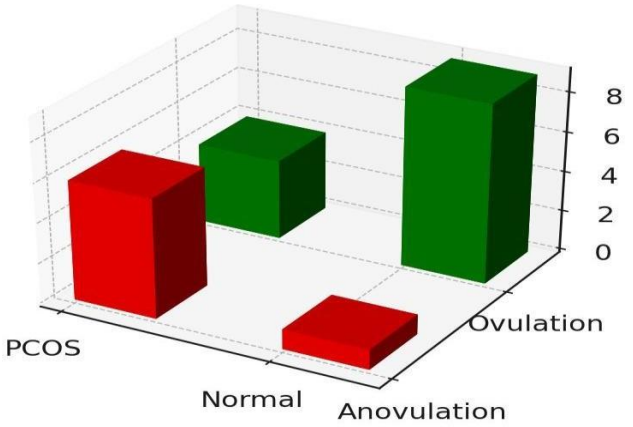
The study revealed a strong association between PCOS and thyroid dysfunction, particularly subclinical hypothyroidism. Among the participants diagnosed with PCOS, a significant proportion (approx. 30–40%) exhibited altered thyroid function, with elevated TSH levels and

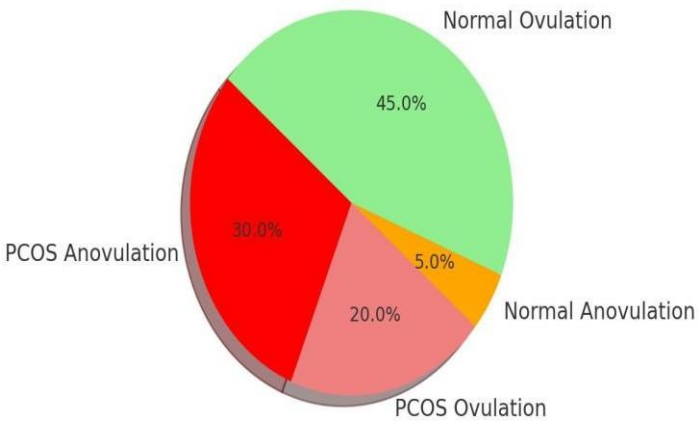
reduced free T4, indicating a potential link between ovarian and thyroid endocrine pathways. Endocrinological assessments showed that patients with concurrent PCOS and thyroid dysfunction had higher levels of insulin resistance (HOMA-IR), elevated serum LH/FSH ratios, and increased androgen levels, particularly testosterone. These hormonal imbalances were more pronounced in individuals who had not adopted lifestyle interventions. Lifestyle management showed a positive impact on endocrine function. Participants who engaged in regular physical activity (at least 150 minutes per week), followed a balanced diet, and practiced stress-reduction techniques (e.g., yoga, mindfulness) demonstrated improved metabolic profiles. This included reductions in BMI, fasting insulin, and serum testosterone levels, along with normalized TSH levels in some cases.

GRAPH-1

GRAPH 1: bargraph on distinguishing between ovulation and anovulation.

GROUP	OVULATION STATUS	PATIENTS
PCOS	Anovulation	6
PCOS	Ovulation	4
Normal	Anovulation	1
Normal	Ovulation	9



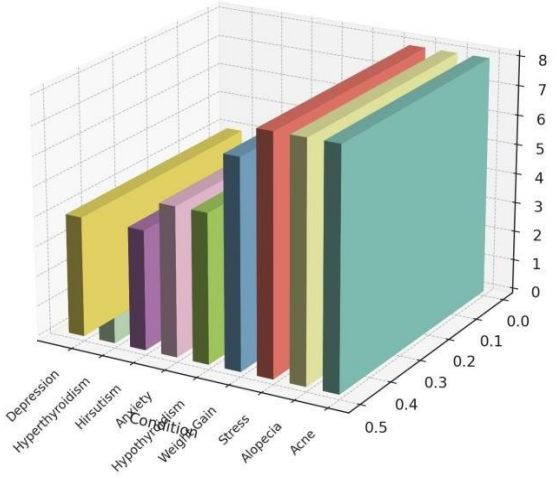


- The first is a bar graph comparing PCOS vs Normal, split by ovulation status.
- The second is a pie chart showing the overall proportion of each group.

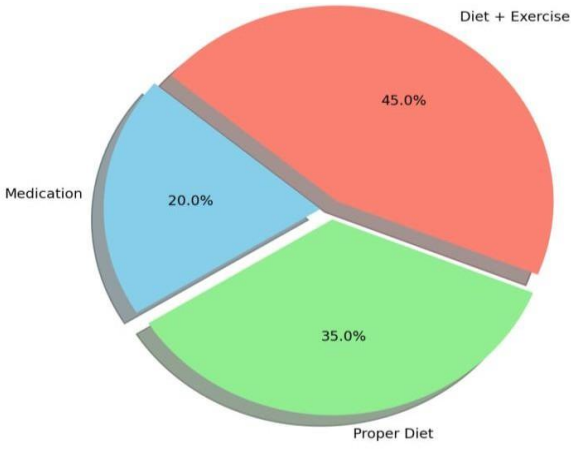
Table 1: Symptoms Of Pcos.

CONDITIONS	PATIENT OUT OF 10
Acne	8
Alopecia (hair thinning)	8
Hypothyroidism	5
Hirsutism	4
Hyperthyroidism	2
Weight gain	7
Stress	8
Depression	4
Anxiety	5

BAR GRAPH ON PREVALANCE OF SYMPTOMS

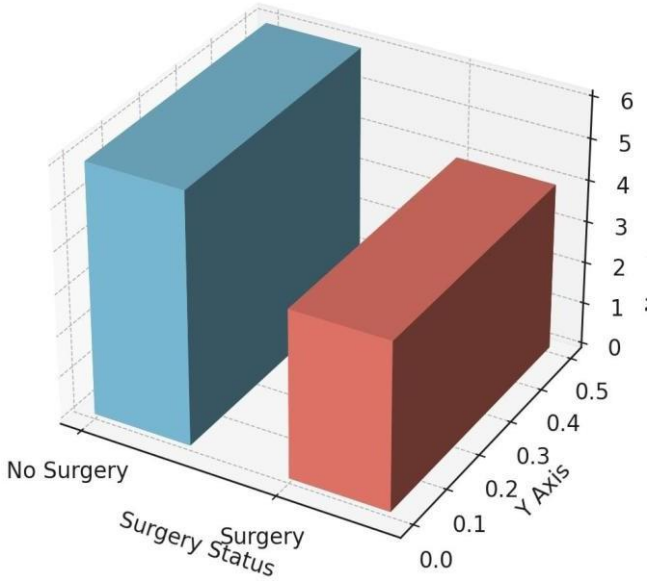


PIECHART ON PCOS TREATMENT



REVIEW ON PIE CHART

- 4 out of 10 can be cured with medication
- 7out of 10 can be cured with proper diet
- 9 out of 10 can be cured with both diet and exercise



Here's the bar graph showing 6/10 individuals who haven't received surgery and 4/10 who have not gone through surgery.

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