

EVALUATION OF REPRODUCTIVE HORMONES (LH & FSH) AND IRON PARAMETERS[HEPCIDINE] AMONG WOMEN DIAGNOSED WITH PCOS

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ABSTRACT

Polycystic ovary syndrome (PCOS) is a complex endocrine and metabolic disorder, typically characterized by LH and FSH levels, iron levels, obesity, insulin resistance, and polycystic ovaries. Lifestyle or diet, environmental pollutants, genetics, gut dysbiosis, neuroendocrine alterations, and iron levels are among the risk factors that predispose females to PCOS. These factors might contribute to upsurging metabolic syndrome by causing ferropenia, hepcidine, 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, Oral iron supplements (e.g., Ferrous Sulfate, Ferrous Fumarate), Lifestyle modification (weight loss, exercise), Letrozole is often the first-line ovulation induction drug PCOS. This study aims to evaluate iron status in women with PCOS and investigate its association with menstrual

irregularities, FSH and LH levels. Assessment of iron status alongside hormonal parameters may provide valuable insights for the management of women with PCOS **Iron status with menstrual irregularities.**

KEYWORDS: Polycystic ovary syndrome, iron status, LH and FSH levels haematological parameters 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 worldwide. 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, 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] 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]

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 iron parameters and hormones like (LH & FSH).

IRON STATUS

This iron overload is primarily linked to chronic menstrual dysfunction (low iron loss) combined with decreased hepcidin levels and insulin resistance, which together increase intestinal iron. Ferritin rises in PCOS patients, especially with abnormal sugar levels, due to high iron in the blood, this high iron is associated with several factors such as menstrual disorders, insulin resistance, and low hepcidin that increases iron absorption, it is not likely that elevated androgens may improve erythropoiesis due to transferrin level do not rise in PCOS women.

Lowered Menstrual Blood Loss (Oligo/Amenorrhea): Because irregular or absent periods mean less blood loss, the body retains iron longer, which raises stored iron (ferritin). Increased Insulin Resistance high circulating insulin^[13] naturally increases intestinal iron absorption and tissue iron deposition. Increased ferritin creates a cycle of oxidative stress that exacerbates insulin resistance Inflammation: Elevated ferritin acts as a biomarker for low-grade chronic inflammation frequently seen in PCOS.^[14]

Ferritin and PCOS

Ferritin is the main iron storage protein in the human body, responsible for sequestering cellular iron reserves to safeguard the cells from the harmful effects of reactive oxygen species, whose formation can be catalyzed by free iron. Ferritin is composed of a total of 24 light chain and heavy chain (FTH) subunits but with different ratios between the 2, depending on the location.^[15] In tissues with a more pronounced iron storage function, such as the liver, ferritin light chain subunits are more prominent, whereas in tissues with more active iron metabolism, such as the skeletal muscles, ferritin heavy chain subunits possessing iron oxidation capability are predominant.^[16] Ferritin is primarily a cytosolic protein, and when cellular iron levels are low, it can be degraded in lysosomes to release its iron content into the cell Ferritin serves as an important regulator not only of iron metabolism but also of redox

balance and energy metabolism via its ferroxidase activity and interplay with mitochondria.^[17] Indeed, knockout of the is embryonic lethal,^[18] and altered ferritin levels have even been implicated in.

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 foetus ready for delivery.^[19] 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 union 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.^[20]

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.

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 hereditary 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 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.^[21]

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.^[22] Hyperandrogenaemia provokes LH excess, which then acts on theca and luteinized granulosa sustaining cycle.^[23]

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.^[24] 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.^[25] This conversion results in chronic feedback at the hypothalamus and pituitary gland, in contrast to the normal fluctuations in feedback.

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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.

Polycystic Ovary Syndrome (PCOS) affects about 8% to 13% of women globally. Of those diagnosed, roughly **50% to 75%** exhibit a characteristic hormonal imbalance where the ratio of Luteinizing Hormone (LH) to Follicle Stimulating Hormone (FSH) becomes elevated^[31] Women with Polycystic Ovary Syndrome (PCOS) commonly experience **mild iron overload** or, conversely, **iron-deficiency anemia**. Iron levels in PCOS are primarily dysregulated by **insulin resistance** and chronic inflammation, which lower *hepcidin* (the hormone regulating iron absorption) and artificially elevate serum ferritin (stored iron).^[32]

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Ferritin and PCOS

Lowered Menstrual Blood Loss (Oligo/Amenorrhea): Because irregular or absent periods mean less blood loss, the body retains iron longer, which raises stored iron (ferritin).

Increased Insulin Resistance: High circulating insulin^[33] naturally increases intestinal iron absorption and tissue iron deposition. Increased ferritin creates a cycle of oxidative stress that exacerbates insulin resistance.

Inflammation: Elevated ferritin acts as a biomarker for low-grade chronic inflammation frequently seen in PCOS.^[34]

Ferritin and PCOS

Ferritin is the main iron storage protein in the human body, responsible for sequestering cellular iron reserves to safeguard the cells from the harmful effects of reactive oxygen species, whose formation can be catalyzed by free iron. Ferritin is composed of a total of 24 light chain and heavy chain (FTH) subunits but with different ratios between the 2, depending on the location.^[35] In tissues with a more pronounced iron storage function, such as the liver, ferritin light chain subunits are more prominent, whereas in tissues with more active iron metabolism, such as the skeletal muscles, ferritin heavy chain subunits possessing iron oxidation capability are predominant.^[36] Ferritin is primarily a cytosolic protein, and when cellular iron levels are low, it can be degraded in lysosomes to release its iron content into the cell. Ferritin serves as an important regulator not only of iron metabolism but also of redox balance and energy metabolism via its ferroxidase activity and interplay with mitochondria.^[37] Indeed, knockout of Fth is embryonic lethal,^[38] and altered ferritin levels have even been implicated in, for example, neurodegenerative diseases. Interestingly, higher ferritin levels have also been associated with metabolic syndrome in population studies.^[39] The serum ferritin levels are correlated with body iron stores under healthy conditions and are, thus, used in clinical practice, with levels between 15 and 200 mg/L for women and 15 and 300 mg/L for men usually considered normal ranges,^[40] the target level being approximately 50 mg/L .^[41] Given that ferritin is an acute-phase reactant, its serum levels increase during inflammation and in certain clinical conditions such as renal failure, which should be considered when analyzing the levels. A commonly suggested reason for low ferritin levels in women is heavy menstrual bleeding (HMB), and in line with this, women with amenorrhea seem to have higher serum ferritin levels given the absence of menses. Polycystic ovary syndrome (PCOS) is the most common endocrine disorder in women of reproductive age, affecting 1 in 8 women, and presents both reproductive and metabolic impairments. The diagnosis of PCOS involves hyperandrogenism (HA; clinical or biochemical), oligomenorrhea or amenorrhea (OA), and polycystic ovarian morphology (PCOM) or excess antimüllerian hormone (AMH) levels. Overweight and obesity are typical facets of the syndrome, affecting up to 70% of women with PCOS, and can further exacerbate other metabolic defects. Polycystic ovary syndrome also commonly includes insulin resistance leading to hyperinsulinemia. This insulin excess enhances androgen production in the ovaries and aggravates the HA, resulting in a cycle of hormonal and metabolic dysregulation. Additional metabolic defects include dyslipidemia, increased blood pressure, and chronic inflammation, all of which can facilitate cardiovascular disease

as well as predispose women with PCOS to type 2 diabetes, and major cardiovascular events. Because PCOS features commonly include OA, women with PCOS often have reduced menstrual blood loss. Consequently, this may result in higher ferritin and hemoglobin (Hb) levels than in women without the syndrome. To support this hypothesis, there is 1 previous study reporting higher Hb levels in women with PCOS in a hospital-based population setting, and some evidence of disrupted regulation of iron metabolism in women with PCOS also exists.^[42] Here, we examined the ferritin levels in women with and without PCOS, and the association of serum ferritin levels with Hb levels, PCOS traits, metabolic profile, and the infertility status. For this purpose, we used the population-based Women's Health Study (WENDY) cohort (23), derived mainly from the Northern Finland Birth Cohort 1986 (NFBC1986), collected in mid adulthood.

- **Lowered Menstrual Blood Loss (Oligo/Amenorrhea):** Because irregular or absent periods mean less blood loss, the body retains iron longer, which raises stored iron (ferritin).
- **Increased Insulin Resistance:** High circulating insulin naturally increases intestinal iron absorption and tissue iron deposition. Increased ferritin creates a cycle of oxidative stress that exacerbates insulin resistance.
- **Inflammation:** Elevated ferritin acts as a biomarker for low-grade chronic inflammation frequently seen in PCOS.

METHODS

Every consecutive female patient in the age group of 18–45 years diagnosed with Polycystic Ovary Syndrome (PCOS) attending the outpatient department of Gynecology and Endocrinology was screened for inclusion in the study.

1. PCOS was diagnosed according to the Rotterdam criteria, which include menstrual irregularities (amenorrhea or oligomenorrhea),
2. clinical and/or biochemical hyperandrogenism,
3. ultrasonographic evidence of polycystic ovaries.

The presence of any two of these three criteria was required for diagnosis after excluding other endocrine disorders. Age-matched healthy women with regular menstrual cycles and no evidence of PCOS served as the control group.

Detailed clinical history, physical examination, and laboratory investigations were performed

for all study participants. Laboratory investigations included estimation of hormonal parameters such as luteinizing hormone (LH), follicle-stimulating hormone (FSH), and the LH/FSH ratio. In addition, iron profile parameters including serum ferritin, serum iron, total iron-binding capacity (TIBC), and Haemoglobin levels were evaluated.

Serum ferritin was measured using enzyme-linked immunosorbent assay (ELISA) and was used as an indicator of body iron stores. Haemoglobin concentration was determined using an automated haematology Analyzer. Serum iron and TIBC were estimated by standard colorimetric methods, and transferrin saturation was calculated from serum iron and TIBC values.

All laboratory investigations were carried out according to standard operating procedures using commercially available diagnostic kits. Data obtained from the study were Analyzed statistically using SPSS [statistical package for the social science] software. Results were expressed as mean \pm standard deviation. Student's t-test and Chi-square test were used for comparison between groups, and a p-value of less than 0.05 was considered statistically significant.

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.

TIBC TEST

Evaluation of iron status is performed using a combination of hematological and biochemical parameters, including hemoglobin, serum iron, serum ferritin, total iron-binding capacity (TIBC), and transferrin saturation.^[43]

A venous blood sample is collected under aseptic conditions, and the serum is separated for biochemical analysis. Hemoglobin estimation is performed to assess the oxygen-carrying capacity of blood and detect anemia. Serum iron measurement determines the amount of circulating iron bound to transferrin in the bloodstream. Serum ferritin is measured to evaluate body iron stores and is considered one of the most reliable indicators of iron reserve. Total iron-binding capacity (TIBC).^[44]

TREATMENT

Strategies for Incorporating Lifestyle Changes to Manage PCOS Symptoms.

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.

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.

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.

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.

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 present study demonstrated that women with polycystic ovary syndrome (PCOS) exhibit

significant alterations in both hormonal and iron profiles when compared with healthy controls. PCOS patients showed elevated luteinizing hormone (LH) levels with relatively reduced or normal follicle-stimulating hormone (FSH) levels, resulting in an increased LH/FSH ratio, which is characteristic of the disorder. In addition, abnormalities in iron metabolism were observed, including increased serum ferritin and serum iron levels, along with changes in total iron-binding capacity (TIBC) and hemoglobin concentrations. These findings suggest a possible association between altered iron homeostasis and hormonal imbalance in PCOS. Therefore, assessment of iron parameters in conjunction with reproductive hormones may provide valuable information for the early diagnosis and comprehensive management of PCOS. Further studies with larger sample sizes are recommended to better elucidate the underlying mechanisms and clinical implications of these alterations.

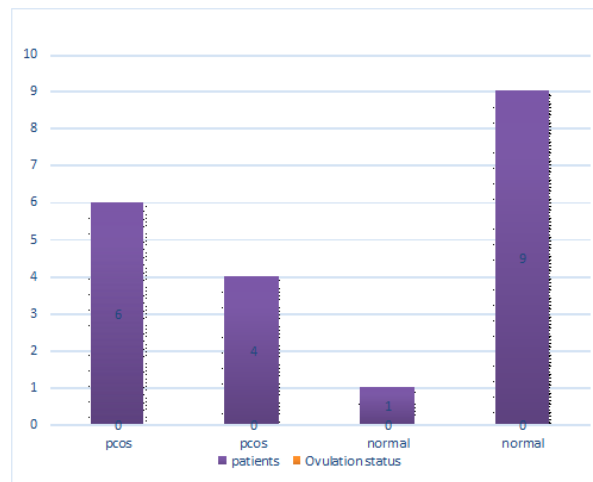
RESULTS

The present study demonstrated a significant association between polycystic ovary syndrome (PCOS), altered iron status, and hormonal imbalance. Women diagnosed with PCOS exhibited significantly higher serum ferritin and serum iron levels compared to healthy controls, suggesting increased iron stores in affected individuals. Hemoglobin levels were also found to be slightly elevated, while total iron-binding capacity (TIBC) showed no significant difference between the groups.

Endocrinological assessment revealed characteristic hormonal disturbances in PCOS patients, including elevated luteinizing hormone (LH) levels and increased LH/FSH ratios, whereas follicle-stimulating hormone (FSH) levels remained within the normal range or were mildly reduced. Correlation analysis demonstrated a positive association between serum ferritin and both FSH and LH levels, with a stronger correlation observed with LH. These findings indicate a possible relationship between iron metabolism and reproductive hormone dysregulation in women with PCOS. Lifestyle interventions, including regular physical activity (at least 150 minutes per week), adherence to a balanced diet, and stress-management practices, were associated with improvements in metabolic and hormonal parameters. Participants following these interventions showed reductions in body mass index (BMI), improved insulin sensitivity, and better regulation of LH and FSH levels, suggesting that lifestyle modification may positively influence iron metabolism and endocrine function in women with PC.

PATIENT GROUP++	OVULATION STATUS	PATIENTS
PCOS	Anovulation	6
PCOS	Ovulation	4
NORMAL	Anovulation	1
NORMAL	Ovulation	9

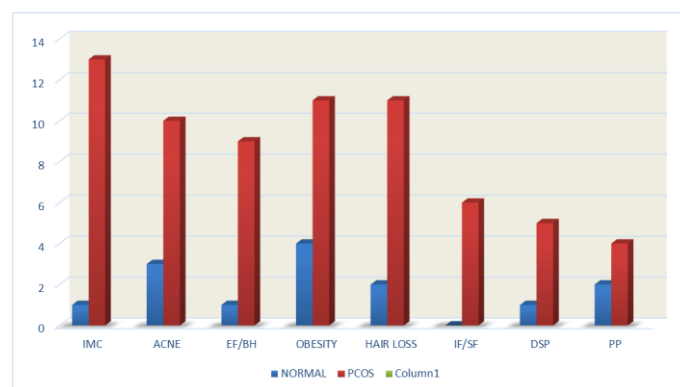
GRAPH 1: Bar graph on distinguishing between ovulation and anovulation.



GRAPH 2.

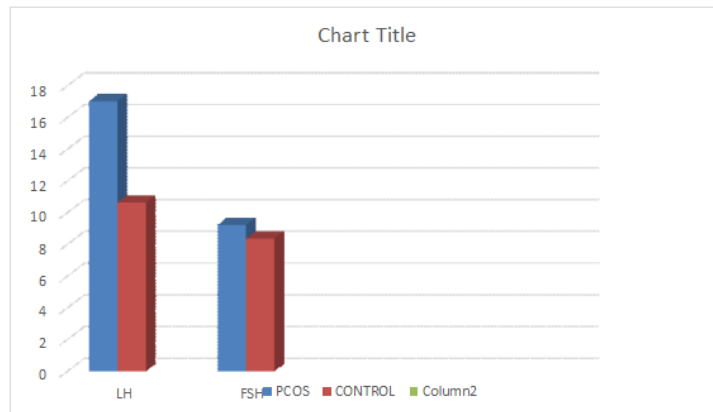
Comparison of Symptoms in Normal Women and PCOS Patients Frequency of common symptoms among 15 normal women and 15 PCOS patients.

Symptoms	Normal	Pcos
Irregular menstrual cycles	1	13
Acne	3	10
Excess facial/body hair	1	9
Weight gain/obesity	4	11
hair loss	2	11
Infertility/subfertility	0	6
Dark skin patches	1	5
Pelvic pain	2	4



GRAPH -3

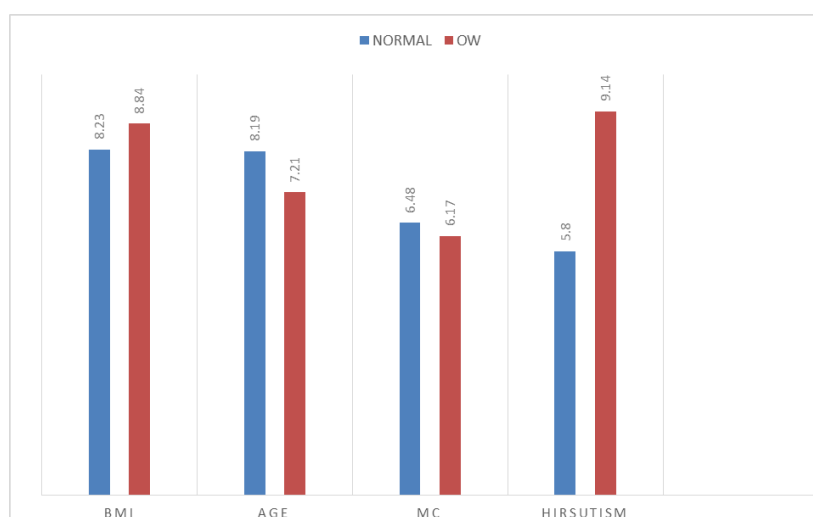
HORMONE	PCOS PATIENTS	CONTROLS	P-VALUE
LH[mIU/mL]	17.02 [16.37-17.83]	10.63[9.49-11.33]	<0.0001
FSH[mIU/mL]	9.22[8.01-10.84]	8.36[7.98-8.99]	0.0765



GRAPH- 4.

hepcidin level in pcos patients depending on some factors.

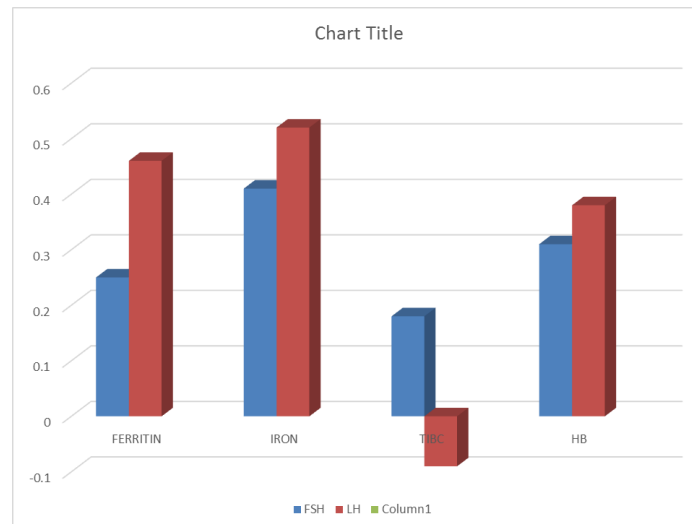
factors	Serum hepcidin		
AGE	Normal 8.23+6.99 [19-29]	Overweight 8.84+8.59 [30-40]	Obese 4.77+2.46
BMI	8.19+8.22	7.21+6.09	
MENSTRUAL CYCLE	Normal 6.48+6.67	Oligoamenorrhea 6.17+3.88	Menorrhagia 9.69+8.78*
HIRSUTISM	With 5.80+4.39	Non	



GRAPH-5.

Comparison of FSH and LH correlation coefficients with iron indices.

PARAMETERS	FSH	LH
Ferritin	0.25	0.46
Iron	0.41	0.52
TIBC	0.18	-0.09
Hb	0.31	0.38



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