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Review

Exercise Benefits During PCOS

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Abstract

A usual endocrine condition that affects women of reproductive age, polycystic ovary syndrome (PCOS) is characterized by irregular menstrual periods, insulin resistance, hormonal imbalance, and polycystic ovaries. Changing one's lifestyle, especially through exercise, is essential for controlling PCOS symptoms and enhancing general health. Regular physical activity has been shown to enhance insulin sensitivity, aid in weight management, regulate menstrual cycles, and reduce androgen levels. Additionally, exercise contributes to improved cardiovascular health, reduced inflammation, and better mental well-being, addressing common comorbidities such as anxiety and depression often seen in PCOS patients. Both aerobic and resistance training yield significant benefits, with a combined approach proving most effective. Incorporating exercise as part of a holistic treatment plan can significantly improve the quality of life and long-term health of individuals with PCOS.

Keywords: PCOS, Exercise, Menstrual cramp, Lifestyle

INTRODUCTION

The condition known as polycystic ovarian syndrome (PCOS), which is also referred to as hyperandrogenic anovulation (HA) or Stein–Leventhal syndrome, is one of the most common endocrine system conditions that affect women of reproductive age. Menstrual disruption, infertility, hirsutism, acne, and obesity are some of the symptoms that can be exhibited by this chronic and diverse illness. It depicts a syndrome in which at least one ovary has an ovarian volume that is larger than 10 millilitres and at least one ovary develops an estimated ten tiny cysts with diameters ranging from 2 to 9 millimetres. When difficulties arise that greatly diminish a patient's quality of life, such as hair loss, alopecia, acne, and issues associated to infertility, it is typically not detected until after the patient has experienced these symptoms. Four to ten percent of women of reproductive age around the world are estimated to have polycystic ovary syndrome (PCOS), according to a comprehensive screening of women that utilised the diagnostic standards of the National Institutes of Health (NIH) [1]. As of 2012, the World Health Organisation (WHO) estimated that 116 million women around the world were affected by polycystic ovary syndrome (PCOS). This high frequency, in addition to its connection with irregularities in ovulation and menstruation, infertility, hair loss, and metabolic disorders, highlights the

considerable financial burden that polycystic ovary syndrome (PCOS) imposes by [2]. The majority of cases of polycystic ovary syndrome (PCOS) are found between the ages of 20 and 30, despite the fact that the condition can manifest at any age, beginning with menarche. 1.55 million women of reproductive age around the world are affected with polycystic ovary syndrome (PCOS), which results in 0.43 million disability-adjusted life years (DALYs). Compared to 2007, the age-standardized incidence rate of polycystic ovary syndrome (PCOS) in women of reproductive age was 82.44 per 100,000 in 2017. This is a 1.45% increase from 2007[3].

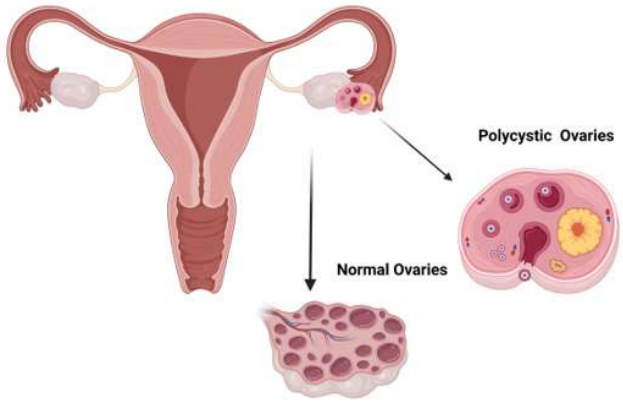

Fig 1: Female reproductive Organ Figure generated with the help of Biorender.com.

On the other hand, it was previously believed that polycystic ovary syndrome (PCOS) was a problem that only affected adult women. However, recent research has shown that PCOS is a syndrome that lasts a lifetime and first becomes apparent during pregnancy. In spite of the fact that the precise reason for this multifactorial disorder is not understood, it is believed that a mix of genetic and environmental variables is the key contributor to the condition. A hormonal imbalance, chronic low-grade inflammation, insulin resistance, and hyperandrogenism are the primary factors that contribute to the pathophysiology of polycystic ovary syndrome (PCOS). These factors have the effect of inhibiting folliculogenesis and increasing the risk of associated comorbidities, such as endometrial cancer and type II diabetes. International recommendations state that hyperandrogenism, ovarian morphology, and anovulation are the three primary variables that are utilised in the process of diagnosing polycystic ovary syndrome (PCOS)[4]. There are a variety of environmental factors that may be contributing to the development, occurrence, and management of polycystic ovary syndrome (PCOS). These factors include geography, diet and nutrition, socioeconomic status, and environmental contaminants such pollution. In the past several years, a connection between polycystic ovary syndrome (PCOS) and the microbiome has been identified, and it is believed that this connection played a role in the development of the illness. Some environmental risk factors may be responsible for dysbiosis of the gut microbial community, which may be a potential pathogenic component in the development and progression of polycystic ovary syndrome (PCOS). Different microbiota are responsible for the origin of distinct pathogenic characteristics of polycystic ovary syndrome (PCOS), and the critical pathways that link their involvement in the onset of various clinical manifestations of PCOS have led to the development of new therapeutic options for the condition [5]. Increasing eubiosis and decreasing the influence of changed microbial profiles are two of the ways that prebiotics, probiotics, synbiotics, and faecal microbiota transplantation (FMTs) can assist in the management of the many phenotypes that are associated with polycystic ovary syndrome (PCOS). It is possible that medicines that are mediated by microbiota could enhance the metabolic, inflammatory, and hormonal features of women who have PCOS.

A summary of the risk factors that may contribute to the formation, prevalence, and modulation of polycystic ovary syndrome (PCOS)is included in this study. Additionally, the paper discusses the potential therapeutic options for PCOS, such as IL-22 and miRNA therapy. In addition, we delve into the significance of gut dysbiosis in the development of polycystic ovary syndrome (PCOS)and assess a number of microbiota-centered therapeutic strategies that have the potential to assist in the management of the illness.[6]

Pathophysiology of PCOS

It is only very recently that it has been recognised that endoplasmic reticulum (ER) stress plays significant roles in the aetiology of a variety of disorders as well as in the maintenance of physiological systems. ER stress is described as a state in which unfolded or misfolded proteins accumulate in the ER as a result of an imbalance between the demand for protein folding and the capacity of the ER to fold proteins. The envelope receptor (ER) is the organelle that is responsible for the folding and assembly of secretory proteins. 49, 53, 57, and 58 are the numbers. The activation of many signal transduction cascades, which are generally referred to as

the unfolded protein response (UPR), is a consequence of ER stress. These cascades have the ability to influence and regulate a variety of cellular activities. As a fundamental principle, the UPR is responsible for restoring homeostasis and maintaining the viability of the cell through three distinct mechanisms. Firstly, it decreases the translation of proteins. Secondly, it enhances the production of ER chaperones, which in turn increases the capacity of the cell to fold proteins. Lastly, it generates ER-associated degradation (ERAD) factors, which eliminate proteins that have been irreparably misfolded. In the event that the stress on the ER cannot be addressed, the UPR will cause the cell to undergo programmed death[7]. A number of pathological disorders that affect humans, such as diabetes, neurodegeneration, cancer, inflammatory conditions, and fibrosis, are under the influence of the ER stress and the UPR, which play important roles in these conditions. 49 and 59 In a manner that is both geographically and temporally coordinated, gonadotrophins and intraovarian factors are responsible for regulating the follicular microenvironment. There are a number of intraovarian factors that play important roles in pathological disorders of the ovary, including polycystic ovary syndrome (PCOS). These factors have regulatory roles throughout the entire process of follicular growth. 39, 53, 60, and 61 are the numbers[8]. For the first time, we were able to demonstrate that ER stress pathways are activated in the granulosa cells of both a mouse model of polycystic ovary syndrome (PCOS) that was caused by the continuous administration of testosterone and in human beings. This finding has been validated by other groups. Numbers 48, 62, 63, 64, 65, 66, and 67 follow. Additionally, we discovered that local hyperandrogenism in the follicular milieu of polycystic ovary syndrome (PCOS) is an activator of endoplasmic reticulum (ER) stress in human granulosa cells, 52 and this discovery has been verified in mouse granulosa cells. 63 Local inflammation and oxidative stress are two additional possible activators of ER stress in the follicular milieu of polycystic ovary syndrome (PCOS). These stressors are strongly associated to ER stress and constitute a vicious circle[9]. Furthermore, the local hyperandrogenic circumstances further activate these pathways. The numbers 45, 49, 50, 51, 52, and 53 PCOS is characterised by an accumulation of AGEs and lipids in the follicular milieu, which may also be responsible for the activation of ER stress or stress. 68 and 69 In the follicular milieu of polycystic ovary syndrome (PCOS), a number of local variables that are amplified may work together to limit ER function, which in turn activates ER stress[10].

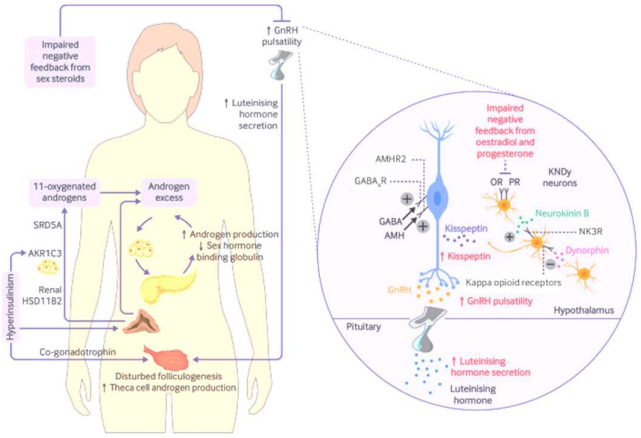

Fig 2: Polycystic ovarian syndrome

Fig 2 Within the context of polycystic ovarian syndrome, the pathophysiology and neuroendocrine disturbance of the hypothalamo-pituitary-gonadal axis are examined. The Left An increase in the pulsatility of gonadotrophin releasing hormone (GnRH) leads to an increase in the secretion of luteinizing hormone, which in turn leads to a disruption in folliculogenesis and an increase in the production of ovarian androgens during the process. Additionally, there is a rise in the levels of adrenal androgens, which include 11-oxygenated androgens. These androgens are activated peripherally by renal 11β-hydroxysteroid dehydrogenase type 2 (HSD11B2) and aldo-keto reductase 1C3 (AKR1C3) in adipocytes. Eleven-ketotestosterone is converted into eleven-ketodihydrotestosterone by the enzyme known as steroid-5α-reductase (SRD5A). By stimulating the accumulation of abdominal adipose tissue, which in turn leads to a rise in insulin resistance and hyperinsulinism, excessive levels of androgens are responsible for the condition. Hyperinsulinism is characterised by the stimulation of AKR1C3 activity, the increase of androgen production from the adrenal cortex and the ovaries (through its role as a co-gonadotrophin), the reduction of hepatic sex hormone binding globulin production, and the inhibition of progesterone-mediated negative feedback onto GnRH neurones, which ultimately leads to a vicious cycle of worsening androgen excess. (That One) Through a combination of paracrine and autocrine mechanisms,

kisspeptin, neurokinin B, and dynorphin A neurones (also known as KNDyneurones) are able to control the release of kisspeptin onto GnRH neurones, which in turn controls the pulsatility of GnRH. In order to induce the release of kisspeptin, neurokinin B binds to neurokinin 3 receptors (NK3R). On the other hand, dynorphin binds to kappa opioid receptors in order to prevent the production of kisspeptin. GABA (gamma-aminobutyric acid) and anti-mullerian hormone (AMH) are two hormones that have the ability to promote the pulsatility of GnRH by binding to GABAA receptors (GABAAR) and AMH receptor type 2 (AMHR2), respectively. At the level of the hypothalamus, there is evidence of impaired negative feedback from oestradiol and progesterone, anomalies in the control of these components that are caused by neuroendocrine dysfunction are shown in red[11]. Oestrogen receptor means "OR," whereas progesterone receptor means "PR." Figure generated with the help of Biorender.com.

A failure of the selection of dominant follicles to ovulate and an ovulatory disorder are the characteristics that define the ovarian dysfunction that is associated with polycystic ovary syndrome (PCOS). This dysfunction is characterised by abnormal follicular growth, which increases in the early stage and stops at the antral stage. 70.0 PCOM with interstitial fibrosis is the morphology of the ovary that is characteristic of persons who have polycystic ovary syndrome (PCOS). 71% We were able to demonstrate that endoplasmic reticulum stress is a contributor to the pathophysiology of polycystic ovary syndrome (PCOS) by demonstrating that it causes several functional abnormalities in granulosa cells (Figure 3). One of the characteristics of polycystic ovarian syndrome (PCOS) is the acceleration of interstitial fibrosis in the ovary, which is accelerated by ER stress. This stress induces the production of transforming growth factor-β1 (TGF-β1), which is a profibrotic growth factor, in granulosa cells. One of the characteristics of polycystic ovary syndrome (PCOS) is the presence of follicular development arrest in the antral stage, which is a result of ER stress. This stress is responsible for mediating the apoptosis of granulosa cells that is triggered by testosterone through the expression of the proapoptotic component death receptor 5 (DR5).[12] Stress on the endoplasmic reticulum (ER) also has a role in mediating the development of receptor for advanced glycation end products (RAGE) in granulosa cells, which leads to the accumulation of AGEs in these cells. The accumulation of 72 AGEs in the granulosa cells of individuals with polycystic ovary syndrome (PCOS) is known to be related with the pathophysiology of the condition. 75 Endoplasmic reticulum stress also activates the aryl hydrocarbon receptor (AHR), which is a typical receptor for extracellular droplets (EDCs), as well as its downstream signalling in granulosa cells. This suggests that it has the potential to modify the steroid metabolism in these cells. 73 Notch signalling, which is one of the most evolutionarily highly conserved signalling systems, is responsible for regulating various cellular processes through juxtacrine cell–cell interactions. Furthermore, ER stress induces the expression of multiple genes that are associated with the expansion of the cumulus oocyte complex (COC) in granulosa cells. Is 76 After conducting measurements on the diameters of COCs, it was shown that the ER stress-notch route is responsible for the growth in the size of COCs[13]. However, it is still uncertain if the hypermaturity of COCs has a causal role in the ovulatory failure that is characteristic of polycystic ovary syndrome (PCOS). 74%

Role of Exercise in Managing PCOS

In the context of physical fitness, aerobic exercise refers to any activity that engages large muscular groups, is performed constantly, and is rhythmic in character. Research has demonstrated that physical activity, and more specifically aerobic exercises, has numerous advantages for the management of PCOS symptoms. These advantages include the enhancement of insulin sensitivity, cardiovascular health, and hormonal equilibrium (figure 3), as well as other benefits [14]. The process by which muscles absorb glucose from the blood is improved through aerobic exercise, which includes activities such as fast walking, jogging, cycling, and swimming. This helps to improve insulin sensitivity. Through a reduction in insulin levels and an improvement in ovarian function, it has the potential to control menstrual periods. A significant reduction in body mass index (BMI) and fat mass was detected in a study on women who had polycystic ovary syndrome (PCOS). Additionally, the levels of follicle stimulating hormone (FSH), free testosterone, and sex-hormone-binding globulin (SHBG) were found to be elevated in the experimental group (exercise for 12 weeks, three sessions of 20 minutes) in comparison to the control group. In addition, the international guidelines for the management of polycystic ovary syndrome (PCOS) recommended that adults between the ages of 18 and 64 should achieve a minimum of 250 minutes per week of moderate-intensity activities or 150 minutes per week of vigorous-intensity activities or an equivalent combination of both, in addition to muscle-strengthening activities (such as resistance and flexibility), ideally on two days per week that are not consecutive. Here is a list of the several forms of aerobic activities that can be used to treat symptoms of polycystic ovary syndrome (PCOS):

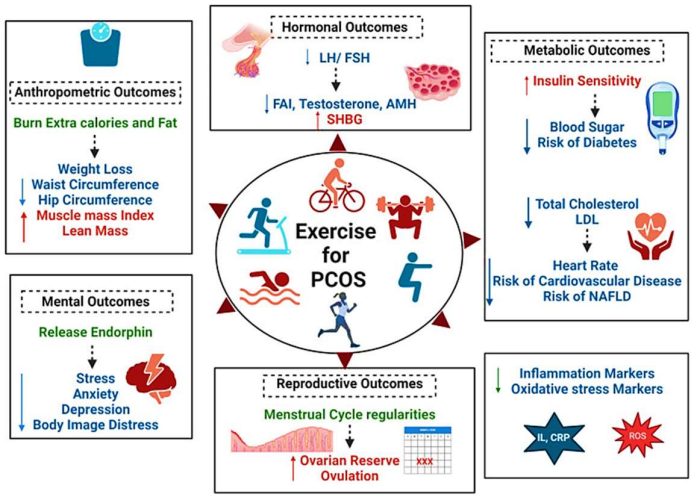

Fig 3: physical activity and exercise

Fig: 3 The influence of physical activity and exercise on the outcomes of anthropometric, hormonal, metabolic, reproductive, and mental health in women who have polycystic ovary syndrome (PCOS). The sign represents a rise, whereas the ↓ sign represents a drop in the consequence that is mentioned adjacent to it. Figure generated with the help of Biorender.com.

Types of Exercise and Their Effects

A polycystic ovary syndrome, often known as PCOS, is an anovulatory disorder that affects between 5 and 7 percent of women who are in the reproductive age range. PCOS is one of the most frequent endocrine illnesses. Among the key pathogenic variables that are present in 55% of women who have polycystic ovary syndrome (PCOS), obesity, insulin resistance (IR), compensatory hyperinsulinemia, sex, and alterations in follicle-stimulating hormone are observed. There is a strong correlation between obesity and insulin resistance, which can result in hyperinsulinemia. This is a characteristic that is frequently observed in women who have polycystic ovarian syndrome (PCOS). In addition to metabolic syndrome and dyslipidaemia, obesity is a contributing factor in the development of cardiovascular disease in women who have polycystic ovary syndrome . In accordance with the guidelines, the first-line treatment for polycystic ovary syndrome (PCOS) is regarded to be the alteration of lifestyle and the management of obesity by diet and exercise. Diet and exercise are highly suggested for women with polycystic ovary syndrome (PCOS) in order to reduce weight, normalise anovulation, and get rid of metabolic syndrome factors. Some of the research provide evidence that physical activity plays a favourable function in the management of insulin resistance in women who have polycystic ovary syndrome (PCOS). The maximal oxygen consumption (MaxVO2), weight, and waist circumferences of patients with polycystic ovary syndrome (PCOS) have been shown to be positively impacted by exercise training, according to data. When it comes to enhancing insulin sensitivity, regulating glycaemic levels, and lowering abdominal fat in obese women who have polycystic ovary syndrome (PCOS), several studies have demonstrated that combined aerobic and resistance training is more effective than either aerobic or resistance exercise alone. Some studies, on the other hand, have indicated that the effects of aerobic and resistance workouts [or diet and aerobic exercise interventions on cardiometabolic health markers in women who have polycystic ovary syndrome (PCOS) are consistent with one another[15].

Aerobic exercise

Recently, and primarily based on research in obesity and type 2 diabetes, lifestyle modification has been accepted as the first line of treatment to address both reproductive and metabolic dysfunctions in women who are overweight or obese and have polycystic ovary syndrome (PCOS). Because of this, there has been a significant increase in the number of clinical trials that have been undertaken that involve food restriction either on its own or in conjunction with physical activity programs. These studies collectively indicate that a reduction in body weight of at least five percent leads to significant improvements in menstrual cyclicity, ovulation, and biochemical hyperandrogenism in terms of reproductive complaints. Additionally, there is an improvement in glucose tolerance and a reduction in the risk of cardiovascular disease. Aerobic exercise led to a 40% higher rate of ovulation (25% versus 65%, respectively) and better changes in SHBG and testosterone despite less weight loss. This was an intriguing discovery that was made in comparison to dietary restriction, which consisted of 800 kcal per day. Aerobic exercise led to a statistically significant decrease in both fasting insulin and insulin resistance

(HOMA-IR), which suggests that there may be a plausible mechanistic connection between the two. It is possible that exercise-induced changes in visceral fat and ectopic lipid in non-fatty tissues are relevant components; however, to the best of our knowledge, there is just one recent study that used single slice computed tomography to measure changes in visceral fat with exercise training in PCOS. The effects of exercise on the polycystic ovarian morphology have not yet been assessed by any studies that have used magnetic resonance imaging or spectroscopy despite the fact that such investigations are uncommon[16].

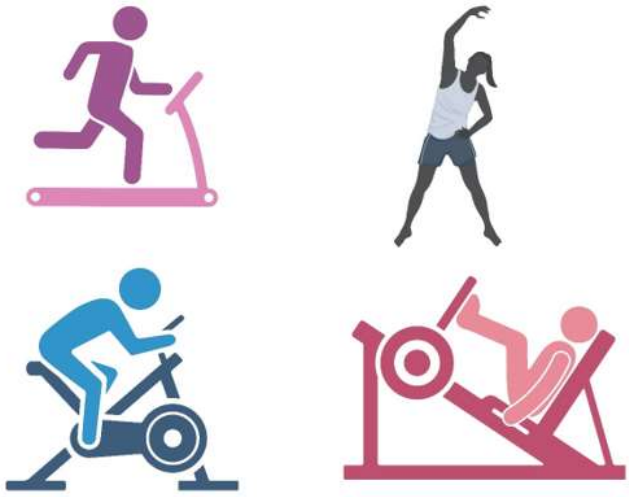

Fig 4: Aerobic exercise Figure generated with the help of Biorender.com.

Resistance training

It is possible that resistance training programs could be good to the health and wellness of women who have polycystic ovary syndrome (PCOS). Additionally, these programs could be a feasible way of exercise for individuals who are deconditioned or unable to endure aerobic exercise. On the other hand, the evidence that has been published to support this assertion is limited. There are only a few primary studies that have been conducted, and these studies often involve small sample numbers (especially randomized controlled trials), heterogeneous sample characteristics, and various degrees of exercise prescription. To further evaluate this body of evidence, additional steps should be taken, such as conducting a systematic review and a meta-analysis. However, based on the potentially positive evidence that was identified from this scoping review, it is evident that there is a need for randomised controlled trials (RCTs) that are rigorously designed, multi-centered, and sufficiently powered. This is necessary in order to determine the certainty of effectiveness and to ensure that the findings can be generalized to the PCOS population as a whole[17].

Fig 5: Resistance training Figure generated with the help of Biorender.com.

High-Intensity Interval Training (HIIT)

This is the first study to explore the effects of high-intensity interval training (HIIT) on anthropometric, lipid profile, insulin sensitivity and resistance, hormonal, and inflammatory indices in PCOS patients. The HIIT regime consisted of 110–100% maximum aerobic volume, four–six sets, and four laps. After a period of eight weeks, the findings demonstrated that high-intensity interval training (HIIT) led to a significant reduction in anthropometric indices, HOMA-IR, LDL, TC, androgenic and inflammatory biomarkers, as well as an increase in insulin sensitivity and aerobic performance. A drop in body weight, fat percentage, waist-to-hip ratio, and visceral fat was observed following eight weeks of high-intensity interval training (HIIT), whereas an increase in VO2max was observed. Moreover, the findings demonstrated that the reduction in visceral fat index, despite the fact that it was seen, was not statistically significant. Hutchison et al. shown that high-intensity interval training (HIIT) was effective in reducing visceral fat. Roessler et al. found that high-intensity interval training (HIIT) decreased weight and waist circumference while simultaneously increasing VO2max in a crossover trial. It was discovered by Almenning et al. in a randomised controlled research that high-intensity interval training (HIIT) and resistance training both decreased body composition and enhanced VO2max. The VO2max was found to be higher in the group that participated in the HIIT.[18] The results of this study are contradicted by the findings of Lionett et al., who found that high-intensity interval training did not enhance fat oxidation. This lack of improvement suggests that PCOS women have metabolic inflexibility. The results of two different meta-analysis studies conducted by Maillard et al. and Santos et al. demonstrated that high-intensity interval training (HIIT) can be utilised as a time-efficient technique to reduce visceral fat and enhance body composition. changes could be attributed to changes in subject characteristics (such as ethnicity, smoking, alcohol consumption, nutritional intake, and physical fitness), as well as differences in the intensity, kind, and length of training being performed. The processes that promote an increase in VO2max are beyond the scope of the current study; nonetheless, it is possible that this is due to an increase in heart output and oxygen heart rate. Over the course of two months of high-intensity interval training (HIIT), Dussin et al. shown that sedentary persons saw an increase in both their cardiac output and stroke volume. The mitochondrial content of many proteins, including citrate synthase, malate dehydrogenase, and pyruvate dehydrogenase, was shown to rise by 18–29 percent after six weeks of participation in high-intensity interval training (HIIT), as reported by Perry et al. As a result of the demand for energy to neutralise protons and boost the regeneration of glycogen and phosphocreatine, you will experience an increase in fat oxidation after performing high-intensity interval training (HIIT).

Fig 6: HIIT Exercise Figure generated with the help of Biorender.com.

Improved Insulin Sensitivity

Research that was published in the journal Fertility & Sterility found that between 65 and 70 percent of women who have polycystic ovary syndrome (PCOS) are insulin resistant. Insulin resistance appears to have a separate link to polycystic ovary syndrome (PCOS), and it is not solely attributable to obesity, even though insulin resistance is more prevalent in obese women, according to the findings of the study. It is Common symptoms of polycystic ovary syndrome (PCOS) include infertility, heavy bleeding, and painful or irregular periods. Among the additional symptoms of polycystic ovary syndrome (PCOS), acne, increased hair growth on the face, weight

gain around the waist, and dark skin spots in the neck, armpits, thighs, and stomach are also present. It is more difficult for women who have high insulin levels to experience these symptoms since they produce more testosterone. This will result in an increase in acne, hair growth, and periods that are erratic or nonexistent. A further point to consider is that women who have insulin resistance are at a greater risk of developing long-term health problems such as diabetes, obesity, cardiovascular disease, and high blood pressure. It is necessary to have excellent symptom management in order to assist your patients in improving their quality of life when they have PCOS. As a result of the fact that PCOS patients are more likely to experience a cycle of rising insulin levels, which can lead to an increase in the desire for carbohydrates and sweets, it is essential to successfully control insulin levels [19].

Hormonal dysfunction and development of PCOS

A significant number of hormonal problems, such as insulin resistance, elevated androgen levels, and disrupted gonadotropin production, are the primary contributors to polycystic ovary syndrome (PCOS). When it comes to the symptoms of polycystic ovary syndrome (PCOS), hyperandrogenism is not only responsible for creating irregular menstrual periods, but it also plays a role in sexual desire and acne. A disruption in ovarian function brought on by gonadotropins that are not properly controlled can lead to the development of follicle cysts. The increase in testosterone production that results from insulin resistance is what initiates the feedback loop. Recent studies have shed light on the intricate hormonal interactions that occur, as well as the significant part that hormonal dysregulation plays in the development and manifestation of polycystic ovary syndrome (PCOS). One of the symptoms of amenorrhoea is the development of cysts in the antral follicles of the ovary, which is caused by an imbalance in hormones. This causes the menstrual cycle to be disrupted and prevents ovulation from occurring. The presence of these cysts, which can develop to a size of up to 10 millimetres and cause the ovaries to extend to a width of up to 10 centimetres, makes it more difficult to become pregnant and prevents fertilisation from taking place. As a result of its association with pregnancy-related disorders such as gestational diabetes and pregnancy-induced hypertension, polycystic ovary syndrome (PCOS) raises the likelihood of glycemia and the birth of babies that are tiny for their gestational age. Theca cells, which are responsible for nourishing growing follicles, respond particularly well to insulin, which causes an increase in androgen levels in PCOS individuals. The disturbance of the hypothalamus's secretion of gonadotropin-releasing hormone (GnRH) is one of the primary hallmarks of polycystic ovary syndrome (PCOS). This disruption disrupts the menstrual cycle and leads to amenorrhoea among women who have PCOS. Secondary amenorrhoea, in which menstrual cycles are absent for three or more regular months, and primary amenorrhoea, in which menarche does not occur, are two types that fall under this condition. Primary amenorrhoea means that menarche does not occur. The presence of an excessive amount of lactotropin hormone has the effect of inhibiting the production of gonadotropin-releasing hormone [20]

CONCLUSION

Exercise serves as a powerful, non-pharmacological intervention in the management of Polycystic Ovary Syndrome (PCOS). It significantly improves insulin sensitivity, reduces androgen levels, aids in weight control, and helps restore hormonal balance. Regular physical activity also has positive effects on mood, reduces the risk of cardiovascular complications, and improves overall life quality. Exercise, whether resistance, aerobic, or a mix of the two, should be seen as an important component of PCOS treatment plans. Educating people with PCOS to start long-term exercise habits can improve disease management and long-term health outcomes.

Future prospects

The improvement of exercise plans for various PCOS symptoms, age groups, and life style situations should be the main focus of future study. Furthermore, incorporating technology like telehealth-based coaching and fitness tracking applications may improve results and adherence. Examining how exercise can work in concert with dietary, pharmaceutical, and mental health treatments could enhance holistic care even further. In order to promote early and sustained lifestyle changes, public health campaigns and educational initiatives should focus on increasing knowledge of the role that physical activity plays in PCOS.

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