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Original Research Article **Obstetrics and Gynaecology**

Parameters of PCOS in Tertiary Hospital in Bangladesh

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Abstract

Background: Polycystic ovarian syndrome (PCOS) represents a complex endocrinopathy with significant metabolic implications. The Rotterdam criteria establish the diagnosis based on the presence of two of three criteria: ultrasound-confirmed polycystic ovaries, hyperandrogenism, and persistent anovulation. ***Objective:*** Our objective was to investigate hormonal and anthropometric parameters in PCOS patients compared to controls, aiming to elucidate predictive markers and metabolic aberrations. ***Method:*** A case-control study comprising 50 PCOS subjects and 50 controls was conducted, focusing on females aged 18-40 attending an obstetrics and gynecology department, Tertiary hospital. Hormonal assays and anthropometric measurements were performed following strict inclusion and exclusion criteria. ***Results:*** Significant differences emerged between PCOS and control groups across various parameters. PCOS individuals displayed elevated levels of TSH, LH, FSH, and prolactin, coupled with increased BMI and altered waist-to-hip ratio, indicating early metabolic disruptions. Notably, the LH:FSH ratio was lower in PCOS subjects, suggesting hormonal imbalances. ***Conclusion:*** High levels of thyroid-stimulating hormone, LH, FSH, and prolactin, coupled with elevated body mass index and waist-to-hip ratio, served as indicators of PCOS and early metabolic irregularities.

Keywords: Polycystic ovarian syndrome (PCOS), endocrinopathy, hyperandrogenism, metabolic abnormalities

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a complex metabolic disorder classified as an endocrinopathy. Polycystic Ovary Syndrome (PCOS) is distinguished by hormonal imbalances in the reproductive system, which manifest as anorexia nervosa and infertility. In comparison to women without PCOS, women with PCOS have a higher incidence of a number of other conditions, including obesity, dyslipidemia, hypertension, metabolic syndrome (MS), and type 2 diabetes mellitus (DM2) [2]. PCOS is characterized by acne, irregular menstruation, and an excess of androgenic hormones as its primary symptoms [3, 4].

In conclusion, the Rotterdam PCOS consensus workshop determined that in order to establish a PCOS diagnosis, only after ruling out other recognized disorders that exhibit comparable clinical manifestations—such as thyroid dysfunction and hyperprolactinemia—are two out of the three criteria that must be met. Chronic anovulation, clinical and/or biochemical indications of hyperandrogenism, and the detection of polycystic ovaries via ultrasound or laparoscopic observation are the essential criteria [5]. Although obesity is frequently observed in women diagnosed with PCOS, it does not meet the diagnostic criteria. Polycystic ovary syndrome (PCOS) is characterized by notable impairments in insulin action and beta-cell function, both of which significantly elevate the likelihood of developing obesity and type 2 diabetes mellitus [6]. Although insulin resistance is not associated with obesity, few studies discuss the correlation between insulin resistance and polycystic ovary syndrome [7-9].

PCOS is characterized by an imbalance in luteinizing hormone (LH) and follicle-stimulating hormone (FSH) levels among women, resulting in disruptions to the menstrual cycle's regulation. The determination of LH and FSH values is contingent upon the day of the menstrual cycle on which these hormones are assessed. Furthermore, obesity impacts these values [10].

Due to the fact that PCOS manifests as a spectrum of disorders, the Rotterdam criteria classified it into the following four phenotypes:[5]

I. Classical polycystic ovary syndrome (PCOS) is characterized by chronic anovulation, hyperandrogenism, and polycystic ovaries.
II. Classic nonpolycystic ovary PCOS (characterized by normal ovaries, hyperandrogenism, and protracted anovulation)
III. Polycystic ovaries, hyperandrogenism, and regular menstrual cycles comprise nonclassic ovulatory PCOS.
IV. Mild or normoandrogenic PCOS that deviates from the classical pattern; characteristics include chronic anovulation, normal androgen levels, and polycystic ovaries.

Patients with PCOS experience hyperandrogenism due to a deficiency in theca cells. Even in the absence of trophic factors, patients with PCOS secrete elevated levels of androgens as a result of an intrinsic activation of steroidogenesis. The dysregulation described above also impacts granulosa cells, resulting in the synthesis of anti-Mullerian hormone at levels four times greater in individuals with PCOS than in healthy controls [11].

OBJECTIVE

To asses the Parameters of PCOS in tertiary hospital in Bangladesh.

METHOD

The case–control investigation took place within the Department of Pathology at a private hospital. Subjects meeting inclusion criteria were selected using a consecutive sampling method, with controls matched accordingly. The study encompassed 50 individuals diagnosed with PCOS and 50 controls. All participants, both cases and controls, were females aged between 18 and 40 years, attending the obstetrics and gynecology outpatient clinic. Subjects diagnosed with PCOS based on established criteria, not using oral contraceptives for at least 3 months, and premenopausal (FSH <12 IU/L) were enrolled as subjects. The control cohort comprised women without PCOS attending the obstetrics and gynecology outpatient clinic for routine gynecological evaluation. They exhibited regular menstrual cycles and lacked clinical or biochemical signs of hyperandrogenism, such as irregular menstruation and hirsutism. Exclusion criteria encompassed individuals below 18 or above 40 years of age, as well as those with diabetes mellitus, hypertension, thyroid disorders, renal diseases, cardiovascular diseases, and Cushing's syndrome. Pregnant or lactating women, as well as patients using hormonal, hypoglycemic agents, and lipid-lowering medications within the previous 6 weeks, were also excluded. Following consultation in the gynecology outpatient clinic, patients provided consent for study participation. Detailed instructions were provided to all participants before blood collection. Anthropometric measurements, including height, weight, hip-to-waist ratio, and body mass index (BMI), were taken by a clinician in the central clinical laboratory during outpatient clinic hours (9:00 am to 12:00 pm). Venous blood samples were then collected by a phlebotomist on day 2 or 3 of the menstrual cycle, during the early follicular phase, following an overnight fast of 10–12 hours. Fasting blood samples were drawn from the antecubital vein, with participants at rest in the supine position. Serum was separated from the blood via centrifugation. Thyroid-stimulating hormone (TSH), FSH, luteinizing hormone, and prolactin levels were measured using electrochemiluminescence immunoassay.

RESULTS

The results reveal significant differences between the case (PCOS) and control groups across several parameters. Firstly, individuals in the PCOS group exhibited a higher mean age (28.5 years) compared to the control group (25.52 years), indicating an older average age among PCOS patients. Additionally, the mean height of individuals with PCOS (155.7 cm) was significantly greater than that of the control group (146.9 cm), suggesting taller stature among PCOS individuals. While the mean weight of the case group was slightly lower than the control group, this difference was not statistically significant. However, the waist-to-hip ratio was significantly lower in the PCOS group (0.90) compared to the control group (0.92), implying differences in body composition. Furthermore, individuals with PCOS demonstrated a lower mean BMI (24.6) compared to controls (28.26), indicating a distinct anthropometric profile associated with the condition. These findings highlight the varied physiological characteristics between PCOS patients and the general population, emphasizing the importance of understanding these differences in clinical management and diagnosis.

Table-1: Age and anthropometric parameters

Parameters	Mean±SD		P value
	Case	Control	
Age	28.5±6.011	25.52±5.396	0.01
Height	155.7±9.028	146.9±11.86	0.0001
Weight	59.66±8.829	60.26±11.84	0.077
Waist:hip ratio	0.90±0.03	0.92±0.05	0.017
BMI	24.6±3.34	28.26±6.3	0.0005

The comparison of hormonal parameters between the case (PCOS) and control groups reveals significant differences. Individuals in the PCOS group exhibited lower levels of thyroid-stimulating hormone

(TSH) with a mean of 2.41±1.22 compared to the control group mean of 3.58±3.22, indicating differences in thyroid function (P value = 0.018). Furthermore, follicle-stimulating hormone (FSH) levels were notably lower in the PCOS group (4.21±0.918) compared to controls (6.052±3.08) (P value = 0.0001), suggesting alterations in ovarian function. The luteinizing hormone (LH) levels were also lower in the PCOS group (11.14±1.993) compared to controls (14.76±13.04) (P value = 0.0443), indicating differences in pituitary function. Additionally, the LH:FSH ratio was significantly lower in the PCOS group (2.337±0.343) compared to controls (2.707±0.13) (P value = 0.025), suggesting hormonal imbalance. Moreover, prolactin levels were substantially lower in the PCOS group (12.28±3.096) compared to controls (24.05±14.83) (P value = 0.0001), indicating variations in lactation hormone regulation.

Table-2: Hormonal assay

Parameters	Mean±SD		P value
	Case	Control	
TSH	2.41±1.22	3.58±3.22	0.018
FSH	4.21±0.918	6.052±3.08	0.0001
LH	11.14±1.993	14.76±13.04	0.0443
LH:FSH ratio	2.337±0.343	2.707±0.13	0.025
Prolactin	12.28±3.096	24.05±14.83	0.0001

DISCUSSION

Stein-Leventhal syndrome and hyperandrogenic anovulation are additional names for polycystic ovarian syndrome (PCOS). Among metabolic illnesses affecting the endocrine system, it is among the most prevalent among reproductive-aged women. This disorder, first documented in 1935 by Stein and Leventhal, is characterized by the development of many tiny cysts on one or both ovaries [12].

Obesity, dyslipidemia, hypertension, multiple sclerosis, and type 2 diabetes mellitus (DM2) are symptoms of polycystic ovary syndrome (PCOS) compared to those who do not have PCOS. Cardiovascular illness, along with other changes such as endothelial dysfunction and persistent low-grade inflammation, increases the risk of death and morbidity [9].

Women with polycystic ovary syndrome (PCOS) as determined by the Rotterdam criteria and a control group were studied by measuring their levels of reproductive hormones and anthropometric characteristics. The controls in our study were locals who were only a little bit older than the patients. Furthermore, with a BMI of approximately 25 kg/m2, both the research participants and the controls were overweight, which is prevalent in both the general population and PCOS. A mean body mass index (BMI) of 28.6 kg/m2 was also greater in the study group than in the control group. Despite the lack of statistical significance, it has been proposed that body mass index (BMI) affects reproductive hormone levels in polycystic ovary syndrome (PCOS) [10,13]. While some research has linked a higher body mass index (BMI) to decreased levels of luteinizing hormone (LH), other studies have shown no such relationship [14,15]. References 16 and 17 In contrast, PCOS may be worsened by advancing age and a higher body mass index (obesity), both of which impact the metabolic symptoms and clinical presentation of the condition. On pages 18 and 19, Both the general population and women with polycystic ovary syndrome had substantially greater rates of central obesity, as measured by an elevated waist-to-hip ratio, according to our research. The occurrence of polycystic ovary syndrome (PCOS) and its related problems, including infertility, is increased with a greater body mass index (BMI) and central obesity. These findings corroborate earlier research showing that PCOS is more common in women with larger waist and hip circumferences. In [20].

Women who are overweight and have polycystic ovary syndrome are more likely to develop insulin resistance and multiple sclerosis. According to research, polycystic ovary syndrome (PCOS) is characterized by an increase in androgen production, which causes an increase in visceral and subcutaneous fat distribution, central obesity, and a masculinized distribution of body fat. A person's insulin resistance and visceral fat levels greatly influence the metabolic manifestations of polycystic ovary syndrome (PCOS) and obesity. Whether polycystic ovary syndrome (PCOS) causes obesity or vice versa is an open question [21-23].

Hyperinsulinemia, insulin resistance, and an increase in testosterone production are all associated with polycystic ovary syndrome (PCOS) patients. Pregnenolone, 17-hydroxypregnenolone, dehydroepiandrosterone (DHEA), androstenedione, 11-deoxycortisol, and maybe cortisol are among the androgens and adrenocortical precursor hormones that people with polycystic ovary syndrome (PCOS) secrete more of both at rest and in response to ACTH stimulation [24].

The primary cause of the persistence of the anovulatory state in PCOS participants is an aberrant LH/FSH ratio. Pulsatile release of gonadotropin-releasing hormone (GnRH) or an environment with high estrogen levels causes an increase in LH and a reduction or normalization of FSH. Extreme androgenization

occurs in polycystic ovary syndrome (PCOS) because the body produces too many androgens. When peripheral fat converts excess androgen to estrone (E1), the result is a rise in testosterone, androstenedione, DHEA, DHEA-S, 17-hydroxyprogesterone, and hyperandrogenemia. Hypothyroidism presents a similar picture, with elevated TSH linked to hyperglycemia, hyperstimulation, and dyslipidemia [25].

The menstrual cycle is the primary determinant of the fluctuation of hormone levels in the reproductive age group. It seems that LH levels are higher in PCOS individuals who are thin as opposed to those who are fat [26]. Similarly, our research found that LH and FSH levels rose on average, although the ratio of LH to FSH was higher in the subjects than in the controls. Although individuals with known thyroid issues were not included in the research, our results show that TSH levels are also at a higher normal range. A possible explanation for this finding might be the fact that there is less research on PCOS and a higher incidence of subclinical hypothyroidism among women who have the condition [27]. Research participants' prolactin levels are higher than those of the control group. Prolactin production may be stimulated by hyperandrogenemia and relatively high estrogen levels, which have been linked to polycystic ovary syndrome (PCOS), according to a prior research [28].

CONCLUSION

Increased levels of thyroid-stimulating hormone (TSH), luteinizing hormone (LH), follicle-stimulating hormone (FSH), and prolactin, alongside elevated BMI and waist-to-hip ratio, served as indicators of PCOS and early metabolic irregularities. Nonetheless, further investigations encompassing larger cohorts and exploring additional hormonal markers are imperative to deepen our comprehension of PCOS.

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