Ranges of Body Mass Index (BMI) in Iraqi Women Patients with Polycystic Ovary Syndrome

Fadaa Abdullah Mahmoud[1]*, Zainab Muayad Saber[2]
[1]College of Health and Medical Techniques, AL-Bayan University, Baghdad, Iraq
Email: fathaa.m@albayan.edu.iq
[2]College of dentistry, AL-Bayan University, Baghdad, Iraq
Email: zainab.m@albayan.edu.iq

Abstract
Objective: To estimate the relationship between polycystic ovary syndrome with weight gain andobesity in asample of Iraqi women through the values of their body mass index. Aim: A comparative, descriptive investigation. Materials and Methods: This study performed at Kamal Al-Samaray Hospital in Baghdad's infertility care and IVF center from August 2022 to October 2022 on totally 80 infertile women aged 18-45 years with clinical characteristics of oligo-/amenorrhea, hirsutism. PCOS patients were identified using the Rotterdam 2003 criteria and SPSS was applied for statistical data analysis. Results: It showed that women with PCOS have differentBMI values. The high percentage of BMI was greater than 30 kg/m2 which refer to obesity, the second BMI was between 25-29.9 kg/m2 which means over weight and the third was between 20 to 24.9 kg/m2 which means optimum weight and finally less than 20 kg/m2 which mean leanness and the differences between the groups are highly significant (P≤0.0001). Conclusions: It is concluded from this study that as compared to women without PCOS, the prevalence of obesity and overweight was substantially greater than optimum weight and leanness.

Keywords: Hirsutism, Oligomenorrhea, Amenorrhea Obesity, PCOS, Iraq

1. Introduction

The mostly prevalent kind of female gynecological endocrinopathy is polycystic ovarian syndrome (PCOS), (1). This syndrome is affecting about 5.6-21.3% of women in reproductive agewho experiences this condition, as a common endocrine disorder (2, 3). PCOS symptoms include amenorrhea or oligoamenorrhoea, hirsutism, obesity, acne, androgenic alopecia, as well as abnormalities of the reproductive system (4). Complex reproductive, metabolic, and psychological characteristics make up PCOS (5, 6). It can be identified by the presence of hypervascularized, androgen-secreting stroma and enlarged ovaries with numerous tiny cysts. Menstrual irregularities, infertility, excessive hair growth, and acanthosis nigricans are further symptoms of this disease (7-10). Anovulation is caused by polycystic ovaries, ovulation-related infertility, amenorrhea, irregular menstruation and excess of androgenic (masculinizing) hormones or their effects. All these etiologies result in hirsutism and acne in addition to insulin resistance, and usually related to high cholesterol levels, Type 2 diabetes, and obesity, which are the main symptoms of this syndrome (7-9). Women who are affected by this syndrome experience varying degrees of symptoms and severity. The clinical method to identify polycystic ovarian syndrome is based on the patient's symptoms, hormone tests, and ultrasound imaging (11, 12). A diagnosis may be aided by certain tests, such as serum levels of prolactin, androgens, testosterone, and androgens. Tests for risk assessment may be recommended to do in patients who have been diagnosed with the syndrome, including endometrial biopsy, pelvic examination, and lipid profile. PCOS exhibits a variety of clinical signs and possible etiologies (8, 9, 13). There is compelling evidence that the condition is inherited. The family clustering of cases, the higher concordance in monozygotic twins compared to dizygotic twins, and the heritability of PCOS's endocrine and metabolic features are a few instances of this evidence (8, 9). A clinical diagnosis of PCOS cannot be made of the condition based on a single diagnostic criterion, according to the 2003 Rotterdam PCOS consensus workshop group funded by ESHRE / ASRM. To fit the definition, two of the three requirements must be satisfied: Infrequent or chronic menstrual periods, polycystic ovaries, and/or clinical and/or biochemical hyperandrogenism (14). Percentage 35% to 80% of PCOS patients has obesity, particularly central visceral obesity, indicating that most PCOS patients but not all-have insulin resistance (15). Endocrine with metabolic disorders are covered up or made more obvious by obesity. Therefore, the purpose of this study is to estimate the link between PCOS and obesity using patient BMI measurements.

2. Materials and methods

Eighty Iraqi women with PCOS aged 18-45 years were recruited from the infertility clinic and IVF center at Kamal Al-Samaray Hospital in Baghdad.

doi.org/10.31838/hiv23.02.1 Received: 05.10.22, Revised: 02.11.22, Accepted: 04.12.22

Based on the Rotterdam Revised Consensus Meeting, diagnostic criteria were utilized to determine the existence of PCOS (14). In addition to body mass index, hormonal measurements, waist-hip ratio (WHR) and the patients' clinical characteristics (oligo-/amenorrhea, hirsutism) were assessed. We evaluated the ovarian morphology using transvaginal ultrasonography. This study focuses on the parameter body mass index and aimed to determine the association between PCOS and obesity according to indicatethe body mass index values that:if BMI is less than 20 kg/m2it mean leanness,if BMI between 20 to 24.9 kg/m2 it means optimum weight,BMI between 25-29,9 kg/m2 means over weight but not reach obesity state and if BMI is greater than 30 kg/m2 it means obesity according to Body Mass Index parameters.

BMI is used as a guide to measure if an individual is of a healthy weight, underweight or overweight. It is calculated by:

Weight (kg) 16-19 = underweight
Height (m2) 20-25 = wholesome weight
26-30 = overweight
31-40 = obese

3. Results and Discussion

The study's findings revealed that women with PCOS have various BMI levels. the high percentage of BMI was greater than 30 kg/m2 which refer to obesity, the second BMI was between 26-29.9 kg/m2 which means over weight and the third was between 20 to 24.9 kg/m2 whichmeans optimum weight and finally less than 20 kg/m2 which mean leanness. These results mean that overweight and obesity were substantially more common than the optimum weight and leanness among PCOS women and the differences between the groups are highly significant (P≤0.0001) as explained in table 1.

Table 1: Distribution of sample study according to Body Mass Index-BMI groups

Percentage (%)	No	BMI groups
40%	32	Obese
33.75%	27	Overweight but donate reach obesity state
25%	20	Optimum weight
1.25%	1	Leanness
100%	80	Total
0.0001	`----	P-value
** (P≤0.01)		
** ≤0.01 level of highly significance		

Obesity and weight gain usually can result in clinical and biochemical expression of PCOS in those females that genetically tend for development of disease, with existence an association between obesity and PCOS as a total 38-88% of PCOS sufferers overweighting or obesity (16-18). BMI and PCOS symptoms are highly associated among different age stages, according to information of the Northern Finland Birth Cohort (NFBC) 1966 (19). Additionally, after slight loss of weight (approximately 5%), clinically substantial changes of metabolic features of PCOS, hyper-androgenic and reproductive are typically seen (16, 20, 21).

Genetic factors have great effect for developing of both PCOS and obesity (22-24). Given the epidemiological connection between the two conditions, it is crucial to take genetic variations' potential roles into account. In UK study, it concluded that there is a strong correlation between the PCOS status, fat mass and obesity-associated gene (FTO: rs9939609 single nucleotide polymorphism), and this association can be weakened through adjusting of BMI between impaired and control cases. These results offered early genetic evidence for the correlation between obesity and PCOS (26). In addition, association between variations in FTO and PCOS status had been verified by later research in a variety of populations (27, 28). According to the study, it's probable that genetic factors like FTO mutations play a part in the onset of PCOS through increasing fat mass and causes weight gain. These genetic effects on fat mass could be likely accounted to some heritability of PCOS.

Numerous epidemiological studies support the strong link between PCOS and obesity (16, 19). Numerous pieces of data show a clear relationship between weight increase and PCOS development and weight loss and PCOS relief, which is often mediated by changes in insulin sensitivity. Additionally, data from the NFBC demonstrate a link between early adiposity rebound in infancy in 1966 and the diagnosis of PCOS and obesity in adults (29). But since PCOS is a complicated disorder, it stands to reason that its connection to obesity is likewise complicated. It's important to consider any potential processes by which PCOS in women with this condition can promote moreover weight gains or thwart successfully the attempting for losing of weight and maintaining body weight through changing of lifestyles. Changing patterns of energy use can cause changes in body weight over time. There is a necessary for considering whether PCOS associated with modifications in a part of metabolism or not. Robinson and colleagues used a cross-sectional strategy to study 14 PCOS-positive and 14 control women using continuous indirect calorimetry. When compared to control ones, postprandial thermogenesis can be much reduced in PCOS women, and even lowered in obese PCOS women (a difference of 41.1 kJ), (30). Additionally, in the PCOS group, the degree of insulin resistance relates to decreasing in postprandial thermogenesis (30).

Obesity affects up to 60% of PCOS patients being examined in the US (31). However, it is less widespread in other populations (32, 33). Additionally, many researchers showed that obesity is low prevalent among adolescents with PCOS symptoms than it is in patients with PCOS who adults in the same community are. Moreover, other researchers have shown that obesity is less prevalent among young people with PCOS symptoms than it is in people with PCOS who adults in the same community (34, 35) are. Therefore, it is illogical to expect that adolescents with PCOS will be as obese as those who have the condition as adults. Visceral obesity is usually seen with a rise in the waist-to-hip ratio of more than 88cm (35 inch) and is connected to several metabolic abnormalities in PCOS, including elevated levels of hyperandrogenemia, insulin resistance, glucose intolerance, and dyslipidemia (36). Finally, obesity is a common finding in women with PCOS, with estimates ranging from 40 to 80 percent of these women being overweight or obese. Additionally, a hereditary propensity to PCOS is strongly supported by the syndrome's familial aggregation (37).

References

Kauffman, R.P., Bake, T.E., Baker, V.M., DiMarino, P. and Castracane, V.D (2008). Endocrine and metabolic differences among phenotypic expressions of polycystic ovary syndrome according to the 2003 Rotterdam consensus criteria. American Journal of Obstetrics &Gynecology, 198, 670.e1-670.e10

Sirmans, S.M. and Pate, K.A(2014). Epidemiology, diagnosis, and management of polycystic ovary syndrome. *Clin. Epidemiol.* 6: 1–13.

Lizneva, D., Suturina, L., Walker, W., Brakta, S., Gavrilova-Jordan, L. and Azziz, R(2016) .Criteria, prevalence, and phenotypes of polycystic ovary syndrome. *Fertil. Steril.* 106: 6-15.

Arshad M, Moradi S, Ahmmadkhani A, Emami Z(2012). Increased prevalence of depression in women with polycystic ovary syndrome. Iranian Journal of Endocrinology and Metabolism; 13: 582-586.

Teede H, DeeksA,Moran L(2010). Polycystic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. BMC Medicine;8:41.

Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Legro RS, et al.(2016). Polycystic ovary syndrome. Nature Reviews Disease Primers;2:16057.

Musmar S, Afaneh A, Mo'alla H(2013).Epidemiology of polycystic ovary syndrome: a cross sectional study of university students at An-Najah national university-Palestine.ReprodBiolEndocrinol. May 20; 11:47.

Legro RS; Strauss JF(2002).Molecular progress in infertility: polycystic ovary syndrome.FertilityandSterility; 78 (3): 569–576.

Kosova G, Urbanek M(2013).Genetics of the polycystic ovary syndrome.Mol Cell Endocrinol. Jul 5;373(1-2):29-38.

Diamanti-Kandarakis E; Kandarakis H, Legro RS(2006).The role of genes and environment in the etiologyofPCOS.Endocrine; 30 (1): 19–26.

Boomsma CM, Fauser BC, Macklon NS(2008)."Pregnancy complications in women withpolycystic ovary syndrome.Semin. Reprod.Med.; 26 (1): 72–84.

Diamanti-Kandarakis E(2010).PCOS in adolescents.BestPract Res ClinObstetGynaecol.; 24(2):173-83.

Dasgupta S, Reddy BM(2008).Present status of understanding on the genetic etiology of polycystic ovary syndrome.J Postgrad Med;54: 115-25.

Rotterdam ESHRE/ASRM(2004).Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long term health risks related to polycystic ovary syndrome (PCOS). Human reproduction.; 19(1): 41-47.

Dale PO, Tanbo T, Vaaler S, Avyholm T(1992).Body weight,hyperinsulinemia, and gonadotropin levels in the polycysticovarian syndrome: evidence of two distinct populations.FertilSteril; 58:487–491.

Barber TM, McCarthy MI, Wass JA, Franks S(2006).Obesity and polycystic ovary syndrome. *ClinEndocrinol (Oxf).*;65:137-145.

Legro RS. The genetics of obesity(2000). Lessons for polycystic ovary syndrome. *AnnNY Acad Sci.*;900:193-202.

18. Balen AH, Conway GS, Kaltsas G(1995). Polycystic ovary syndrome: the spectrumof the disorder in 1741 patients. *Hum Reprod.*;10:2107-2111.

Ollila MM, Piltonen T, Puukka K, et al(2016). Weight gain and dyslipidemia in early adulthood associate with polycystic ovary syndrome: prospective cohort study. J ClinEndocrinol Metab.;101:739-747.

Kiddy DS, Hamilton-Fairley D, Bush A, et al(1992). Improvement in endocrine and ovarian function during dietary treatment of obese women with polycystic ovary syndrome. ClinEndocrinol (Oxf).;36:105-111.

Holte J, Bergh T, Berne C, Wide L, Lithell H(1995). Restored insulin sensitivity but persistently increased early insulin secretion after weight loss in obese women with polycystic ovary syndrome. J ClinEndocrinol Metab.;80:2586-2593.

Walley AJ, Blakemore AI, Froguel P(2006).Genetics of obesity and the prediction of risk for health. *Hum Mol Genet.*;15 Spec No 2:R124-R130.

Franks S. Polycystic ovary syndrome(1995). N Engl J Med.;333:853-86.

Barber TM, Franks S(2013). Genetics of polycystic ovary syndrome. Front Horm Res;40:28-39. .

Day F, Karaderi T, Jones MR, et al(2018). Large-scale genome-wide meta-analysis ofpolycystic ovary syndrome suggests shared genetic architecture for different diagnosis criteria. Plos Genet.;14:e1007813.

Barber TM, Bennett AJ, Groves CJ(2008). Association of variants in the fat massand obesity associated (FTO) gene with polycystic ovary syndrome. *Diabetologia.*;51:1153-1158.

Li T, Wu K, You L, et al. (2013). Common variant rs9939609 in gene FTO confers risk to polycystic ovary syndrome. *PLoS ONE.*;8:e66250.

Yan Q, Hong J, Gu W, et al. (2009). Association of the common rs9939609 variant of FTO gene with polycystic ovary syndrome in Chinese women. Endocrine.;36:377-382.

Koivuaho E, Laru J, Ojaniemi M, et al.(2019). Age at adiposity rebound in childhood isassociated with PCOS diagnosis and obesity in adulthood-longitudinal analysis ofBMI data from birth to age 46 in cases of PCOS. Int J Obes (Lond).;43: 1370-1379.

Robinson S, Chan SP, Spacey S, Anyaoku V, Johnston DG, Franks S.(1992). Postprandial thermogenesis is reduced in polycystic ovary syndrome and is associated with increased insulin resistance. ClinEndocrinol (Oxf).;36:537-543..

Azziz R, Sanchez LA, Knochenhauer ES, et al.(2004).Androgen excess in women: experience with over 1000 consecutive patients. J ClinEndocrinol Metab;89:453–462.

Carmina E, Koyama T, Chang L, Stanczyk FZ, Lobo RA.(1992).Does ethnicity influence the prevalence of adrenalhyperandrogenism and insulin resistance in polycystic ovary syndrome? Am J ObstetGynecol;167:1807–1812.

Carmina E, Legro RS, Stamets K, Lowell J, Lobo RA.(2003).Difference in body weight between American and Italian womenwith polycystic ovary syndrome: influence of the diet. Hum Reprod;18:2289–2293.

Gülekli B, Turhan Nض, Senِz S, Kukner S, Oral H, Gokmen O. (1993).Endocrinological, ultrasonographic and clinical findingsin adolescent and adult polycystic ovary patients: a comparative study. GynecolEndocrinol;7:273–277.

Dramusic V, Goh VHH, Rajan U, Wong YC, Ratnam SS. (1997).Clinical, endocrinologic, and ultrasound features of polycysticovary syndrome in Singaporean adolescents. J PediatrAdolesc;10:125–132.

Ehrmann DA(2005). Polycystic ovary syndrome. N Engl J Med;352:1223–1236.

Sam S, Dunaif A.(2003).Polycystic ovary syndrome: syndrome XX? Trends Endocrinol Metab;14:365– 370.