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Does body mass index have an effect on the prevalence of various symptoms of polycystic ovary syndrome and their associated risk factors?

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Abstract: The present cross-sectional study is an attempt to understand the effect of body mass index (BMI) on the prevalence of various symptoms of polycystic ovary syndrome (PCOS) and to evaluate its associated risk factors.

A total of 250 PCOS women diagnosed by Rotterdam Criteria (2003), age ranging from 18–45 years, attending OPD of Gynaecology and Obstetrics of PGIMER, Chandigarh, India were enrolled in the study. All the participants were divided in three groups according to their body mass index (BMI).

The polycystic ovaries (83.2%) were the most frequently occurring symptoms of PCOS followed by hirsutism (74.4%), oligomenorrhea (60%), seborrhea (45.2%) and acne (40%). Category wise frequency distribution showed higher prevalence of symptoms among women in overweight/obese category, which were further supported by correspondence analysis. Results of multivariate analysis revealed that marital status, type of diet, socio-economic status and physical activity level were potential risk factors contributing to severe manifestations of PCOS symptoms.

Obesity denoted as an important risk factor can exaggerate many symptoms of PCOS and also be a causative factor for menstrual disturbance.

Key words: Body mass index, hirsutism, hyperandrogenism, menstrual disturbance, polycystic ovary syndrome

Introduction

Polycystic ovary syndrome (PCOS) is the most common gynaecological endocrinopathy among women in reproductive age, affecting 5–20% of women worldwide (Azziz et al. 2016). The reproductive characteristics of PCOS includes excessive production of androgens, dysfunctionality of gonadotropin secreting hormones leading to menstrual disturbance, hirsutism and infertility (Sam and Dunaif 2003). The exact pathophysiology of PCOS is still poorly understood

Original Research Article Received: August 29, 2020; Revised: May 15, 2021; Accepted: May 17, 2021 DOI: 10.2478/anre-2021-0013 © 2021 Polish Anthropological Society and characterized with varying manifestations of hyperandrogenism, ovulatory dysfunction and polycystic ovary morphology (Ekwutosi et al. 2012). The clinical and biochemical features of PCOS women may differ according to ethnicity and different criteria used for its diagnosis (Kauffman et al. 2002).

It was observed by previous studies (Balen et al. 1995; Liou et al. 2009) that obesity exacerbates the clinical and biochemical features of polycystic ovary syndrome (PCOS). Diamanti-Kandarakis (2007) illustrated that the effect of obesity on the symptoms of reproductive functions in PCOS is likely to be intermediated by insulin resistance. A variation in the clinical features of PCOS women has been noticed with a higher proportion in obese PCOS women in comparison to their age matched normal weight PCOS women (Gambineri et al. 2002) .Considerable literature (Franks et al. 1991; Holte et al. 1994; Bernasconi et al. 1996; Hsu 2015; Ahmadi et al. 2017) has noted that obese PCOS women presented severe functional hyperandrogenism and a close correlation may exist between adiposity and severity of symptoms in PCOS women. Conflicting evidence was recorded in some studies that subgroups with respect to body mass index did not affect the clinical features of women with polycystic ovary syndrome (Liou et al. 2009; Tamimi et al. 2009). Therefore, consistent evidence documenting enhanced body weight may favor a more severe clinical presentation of symptoms in PCOS. Consequently, the present study was conducted with the primary objective; (a) to determine the impact of body mass index in the clinical presentation of PCOS women and; (b) to evaluate the possible risk factors associated with the progression of PCOS.

Material and methods

The present cross-sectional study was comprised of 250 PCOS women with age ranging from 18 years to 45 years. Data were collected from the patients who visited the Out-Patient Department (OPD), Department of Obstetrics and Gynecology in Postgraduate Institute of Medical Research and Education (PGIMER), Chandigarh, India.

Inclusion criteria: Participants who fulfilled the following criteria were enrolled in the study:

- 1. A participant must be aged between 18–45 years.
- 2. A participant must live in the Chandigarh Capital Region.
- 3. A participant follow the Rotterdam Criteria for PCOS diagnosis.

Exclusion criteria: Any subject having congenital adrenal hyperplasia, androgen secreting tumors (hypothyroidism) or taking any medication influencing the endocrinal parameters of PCOS were excluded from the study.

Our research design enrolled only those patients from the Out-Patient Department (OPD), of (PGIMER) who fulfiled our inclusion criteria and were identified with PCOS on the basis of Rotterdam Criteria (2003). The estimated prevalence of PCOS in North India is 3.7% (Gill et al. 2013). The sample size was assessed using the following formula with 95% of confidence interval and 5% probability of type 1 error (Charan and Biswas 2013);

$$n = Z\alpha^{2} \times p \times q / d^{2}$$
(1.96)² × 3.7 × 0.96 / (0.05)²

where:

 $Z\alpha = 1.96$ for 95% level of confidence; α – the risk of type 1 error; p - expected prevalence;

q – 1 – p;

d – the error of estimate.

The calculated sample size was 50 women by using the above mentioned formula.

Date of birth of each subject was asked and then it was converted into decimal age following decimal age calendar of Tanner et al. (1966). Additional information on age of menarche, marital status, occupation, diet (vegetarian, non-vegetarian and ovatarian) as well as frequency of food intake (once, twice or thrice/day) was recorded as per interview schedule. Each subject was asked when menarche had commenced using menarche recall method. Then Kalpan Meier Survival Curve analysis was employed to estimate their age at menarche.

According to Rotterdam Criteria (2003) a total of two out of following three symptoms i.e. oligo-and/or anovulation (defined by the presence of oligomenorrhea or amenorrhea); clinical and/or biochemical signs of hyperandrogenism (defined by presence of hirsutism (Ferriman-Gallwey score ≥ 6), acne or alopecia, and/or elevated androgen levels); and polycystic ovaries by gynecological ultrasound are required for the diagnosis of PCOS. Clinical hirsutism is defined by the presence of hirsutism, acne or androgenic alopecia. Hirsutism was defined by Ferriman-Gallwey score ≥ 6 . The assessment of m-FG score was determined by using density of terminal hair at nine different body regions, i.e. upper lip, chin, chest, upper back, lower back, upper abdomen, lower abdomen, arm and thigh. The presence of acne was diagnosed by the presence of lesion on the face, back and chest. Presence of oily skin and family medical history of PCOS and father's hair growth i.e. normal, excessive or

baldness was recorded during personal interviews. Detailed menstrual history was also recorded using FIGO classification (Munro et al. 2018) for abnormal uterine bleeding. Normal menstrual frequency was considered if periods ranged from \geq 24 to \leq 38 days, oligomenorrhea if >38 days and polymenorrhea if <24 days. Polycystic ovary morphology was ascertained by following ASRM/ESH-RE criteria and considered to be polycystic ovaries if ovarian volume were \geq 10cm³(Balen et al. 2003).

The height (cm) of the participants was measured by an anthropometric rod with a precision of 0.1mm. Weight (kg) was measured with weighing scale with a precision of 1.0 kg. These anthropometric measurements were taken without shoes and with minimal clothing. BMI was calculated using formula weight (kg)/height (m²). All PCOS women were divided in three groups according to BMI (kg/m²) categories cut-off values for Asian population (WHO 2004) i.e. underweight (BMI < 18.50), normal weight (BMI = 18.50–23.0) and overweight/ obese (BMI>23).

Physical activity was determined using MPAQ scale (Anjana et al. 2015) with self-reported activities of the previous week. Total energy expenditure (TEE) had been calculated by factorial calculation recommended by joint FAO/UNU/ WHO (2001). Minutes spent in each activity was multiplied by physical activity ratio (PAR) to derive TEE of 24 hours, while energy cost was reported as multiple of basal metabolic rate (BMR). Physical activity level (PAL) was estimated by TEE/BMR for 24 hours and divided into 3 categories i.e sedentary (1.40–1.69), moderately active (1.70–1.99) and vigorously active (2.00-2.40).

The socio-economic status of each participant was gauged on the basis of 22 items using Agarwal Scale (2005) and divided participants into High, Upper-middle, Lower-middle, Poor categories.

Statistical package for the social sciences (SPSS) version 19.0 (SPSS Inc.) was used for all statistical analysis and p values <0.05 at confidence interval of 95% were considered statistically significant for all the models. The quantitative data was expressed as mean \pm SD, while the qualitative data as frequency and percentages. The statistical significance in the differences of underweight, normal weight and overweight/obese categories was estimated by chi-square test. Correspondence Analysis (CA) is a two-dimensional multivariate graphical presentation to determine the relationship between various symptoms associated in PCOS women with respect to BMI categories. Univariate regression analysis was used to find out the possible risk factors associated with progression of PCOS. Furthermore, those variables which showed statistically significant association in univariate regression model were investigated in the multivariate model.

The study was approved by the Institutional Ethical Committee of Panjab University, Chandigarh (PUIEC/2018/109/A/09/01), as well as the Ethical Committee of Postgraduate Institute of Medical Research and Education (PGIMER), Chandigarh (INT/ IEC/2018/000450). A written informed consent form was also signed by each participant before enrolling in the study.

Results

The baseline characteristics of PCOS women are presented in Table 1. The

mean age at menarche in the female of present study was 13.62±1.71 years as assessed by Kalpan Meier Survival Curve. The socio-demographic profile showed that 59.6% women were unmarried, while a substantial proportion of women (73.2%) had graduated or had achieved a higher educational level. Most participants (40%) were not employed, while approximately 35.6% were students. The socio-economic status as evaluated by employing Agarwal scale showed that most women came from the upper-middle class (61.6%), followed by lower-middle (28.4%) and high class (8.8%). A considerable proportion of PCOS women were overweight/obese (52.8%), whereas 43.6% of women had normal weight, while 3.6% were underweight . The physical activity level demonstrated that 49.6% PCOS women were moderately active and 47.6% had sedentary lifestyle, while a very small proportion of PCOS women (2.8%) had a vigorously active lifestyle. Table 2 shows the overall, as well as the category wise prevalence of various symptoms associated with PCOS females of the present study.

Out of the total symptoms, polycystic ovaries (83.2%) was the most frequently occurring symptom followed by hirsutism (74.4%), oligomenorrhea (60%), seborrhea (45.2%) and acne (40%). Regarding father's hair growth, approximately 25.6% of fathers of PCOS females exhibited excessive hair growth, while 68% had moderate hair growth and 6.4% fathers showed baldness.

Category wise frequency distribution of symptoms demonstrated that hirsutism was significantly higher (chi-square -6.8, p<0.05) in overweight/obese PCOS women (42%) than women with normal weight (30.8%) and underweight category (1.6%). Prevalence of partici-

Baseline characteristics	Total (n=250)	Chi-square <i>p</i> -values	
Age at Menarche ¹	13.62 ± 1.71	_	
Marital status ²			
Unmarried	149* (59.6%)	< 0.01	
Married	101 (40.4%)		
Education level ²			
Illiterate	2 (0.8%)		
Primary to matric	18 (7.2%)	< 0.001	
High school	47 (18.8%)		
Graduation or Higher	183* (73.2%)		
Occupation ²			
Working	61 (24.4%)	< 0.01	
Non-working	100* (40%)	< 0.01	
Students	89 (35.6%)		
BMI category ²			
Underweight	9 (3.6%)	-0.001	
Normal	109 (43.6%)	< 0.001	
Overweight/Obese	132* (52.8%)		
Socio-economic Status ²			
High	22 (8.8%)		
Upper-middle	154* (61.6%)	< 0.001	
Lower-middle	71 (28.4%)		
Poor	3 (1.2%)		
Physical activity Level ²			
Sedentary	119 (47.6%)	<0.001	
Moderately Active	124* (49.6%)	< 0.001	
Vigorously active	7 (2.8%)		

Table 1. Baseline characteristics of PCOS women under study

¹Continuous variable represented as mean±S.D, ²Categorical variable represented as n (%).

pants with oily skin (Seborrhea) was also highest in the overweight/obese (24.4%) category, followed by normal weight category (19.6%) and negligible percentage (1.2%) were in underweight category. However, these differences lacked statistical significance (p>.05) as it is clear from their chi-square value. Approximately 36% fathers of overweight/obese PCOS participants demonstrated moderate hair growth and 14.8% had excessive hair growth, while baldness (3.2%) exhibited the highest frequency in the fathers of normal weight subjects.

Out of the total subjects, only 21.2% participants had a family history of PCOS, 11.6% of which were from the

overweight/obese category, while 8.8% and 0.8% were from normal weight and underweight categories respectively. A considerable proportion (60%) of PCOS females did not show the presence of acne, it was recorded in 40% females only. The frequency of presence of acne was highest in the overweight/obese category (22%) and results of chi square revealed non-significant (p>0.05) difference in all three groups of body mass index. In the present study, 83.2% of PCOS women had polycystic ovaries (ovarian volume $\geq 10 \text{ cm}^3$) as determined by following ASRM/ESHRE criteria. It was noted that ovarian volume $\geq 10 \text{ cm}^3$ was highest among overweight/obese PCOS women (44%) than normal weight (36%) and underweight (3.2%) categories body mass index, while the differences were statistically non- significant.

A significant proportion of PCOS women (79.6%) exhibited oligomenorrhea (>38 days), while 15.2% of PCOS women had a normal menstrual cycle, and 5.2% of women had polymenorrhea. Based on Table 2 the maximum PCOS women with oligomenorrhea were in overweight/obese category (42.4%,) followed by normal weight (34.4%) and underweight (2.8%). Women with normal menstruation cycle also presented a high frequency in overweight/obese group (8.8%), however, 5.6% women had normal weight, while negligible percentage of women were underweight (0.8%). The frequency of overweight/obese women with polymenorrhea was 1.6%, whereas 3.6% women had normal weight. The overall prevalence of clinical hyperandrogenism (i.e. hirsutism, acne, seborrhea and menstrual irregularity) was higher in overweight/obese PCOS women as compared to their normal weight and underweight PCOS counterparts.

Various symptoms of PCOS women categorized on the basis of their BMI were graphically presented by employing Correspondence Analysis (Fig. 1). The two dimensional plot indicated that overweight/obese PCOS women were associated with symptoms of clinical hyperandrogenism (i.e. hirsutism,

Symptom	s of PCOS	Underweight N = 09 N (%)	Normal N = 109 N (%)	Overweight/ obese N = 132 N (%)	Total N = 250 N (%)	Chi- square
Hirsutism (Clinical hyper- androgenism)	Present	4 (1.6%)	77 (30.8%)	105* (42%)	186 (74.4%)	
	Absent	5 (2%)	32 (12.8%)	27 (10.8%)	64 (25.6%)	6.88*
Oily skin (Seborrhea)	Present	3 (1.2%)	49 (19.6%)	61 (24.4%)	113 (45.2%)	0.56
	Absent	6 (2.4%)	60 (24%)	71 (28.4%)	137 (55.8%)	0.50
Father hair growth	Excessive	1 (0.4%)	26 (10.4%)	37 (14.8%)	64 (25.6%)	
	Moderate	5 (2%)	75 (30%)	90* (36%)	170 (68%)	13.14**
	Baldness	3 (1.2%)	8 (3.2%)	5 (2%)	16 (6.4%)	
Family history of PCOS	Present	2 (0.8%)	22 (8.8%)	29 (11.6%)	53 (21.2%)	0.12
	Absent	7(2.8%)	87 (34.8%)	103(41.2%)	197 (78.8%)	
Acne	Present	3 (1.2%)	42 (16.8%)	55 (22%)	100 (40%)	0.41
	Absent	6 (2.4%)	67 (26.8%)	77 (30.8%)	150 (60%)	0.41
Maximum Ovarian volume	<10 cm ³	1 (0.4%)	19 (7.6%)	22 (8.8%)	42 (16.8%)	0.24
	$\geq 10 \text{ cm}^3$	8 (3.2%)	90 (36%)	110 (44%)	208 (83.2%)	0.24
Menstrual frequency (in days)	Polymenorrhea (<24 days)	0	9 (3.6%)	4 (1.6%)	13 (5.2%)	
	Normal (24–38 days)	2 (0.8%)	14 (5.6%)	22 (8.8%)	38 (15.2%)	4.50
	Oligomenorrhea (>38 days)	7 (2.8%)	86 (34.4%)	106 (42.4%)	199 (79.6%)	

Table 2. Frequency distribution of various PCOS symptom with respect to their body mass index (BMI)

oily skin (seborrhea), oligomenorrhea (irregular menstruation) and excessive father's hair growth), whereas normal weight PCOS women were associated with absence of acne, no history of PCOS and absence of hirsutism. Underweight PCOS women showed a close relationship with baldness in their fathers and normal menstruation.

The univariate regression analysis was performed to assess the possible determinants for the development of PCOS (Supplementary data 1). Those variables which had shown statistically significant association in the univariate model were further investigated in the multivariate regression model.

According to multivariate regression analysis (Table 3) unmarried women had 2.7 times (95% CI 1.1–6.7, p<0.05) more probability to have hirsutism in reference to married women. Women who were consuming a vegetarian diet had 2.3 times (95% CI 1.0–5.5, p<0.05) more risk to develop seborrhea in reference to PCOS women on an ovatarian diet. High class women had 7.3 times (95% CI 2.3–22.8, *p*<0.01) more probability to be associated with hirsutism, women with upper-middle class had 1.1 times (95% CI 0.5–2.1, *p*<0.05) greater risk to experience hirsutism, while lower-middle women had 1.0 times (95%) CI 0.1–1.1, *p*<0.05) greater chances for the same. High class women had 8

Symmetrical Normalization

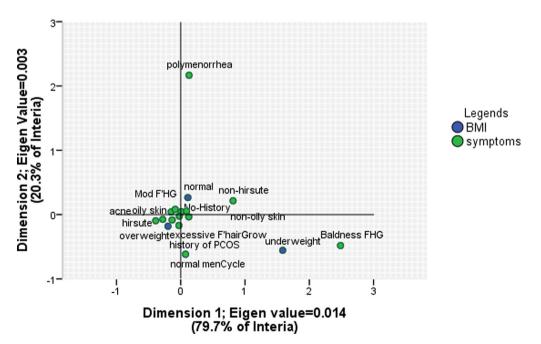


Fig. 1. Correspondence Analysis showing various symptoms of PCOS women categorized on the basis of their body mass index

times (95% CI _2.4–26, p<0.01) higher possibility to develop seborrhea. Upper-middle class were 2.9 times (95% CI _1.6–5.4, p<0.01) more risk to experience oily skin, however, lower-middle class women were 1.8 folds (95% CI _1.8–18.4, p<0.05) more likely to have the same symptoms. Additionally, high class women were 3.4 times (95% CI_1.8–10.1, p<0.01) more likely for the occurrence of acne than the reference population. Additionally, Upper-middle class women had 3.1 times (95% CI _1.6–5.7, p<0.01) more likelihood for

developing acne, however, lower-middle class women had 1.8 times (95% CI _0.1–1.8, p<0.05) higher probability for the occurrence of acne. High class women exhibited 9 times (95% CI_0.7–11.4, p<0.01) more likely to have polymenorrhea (frequent menstruation), whereas upper-middle class women had 5.5 times (95% CI_0.9–31.7, p<0.01) and lower-middle class women presented 1.3 (95% CI_0.1–1.3, p<0.05) greater risk to have polymenorrhea than poor class PCOS women. Participants with sedentary lifestyle presented 5.5 times (95%

Table 3. Multivariate regression analysis for symptoms associated with PCOS women with respect to socio-demographic profile, diet and physical activity level

Variables	Hirsutism	Seborrhea	Acne	Menstrual Frequency OR (95% CI)		
	OR (95%CI)	OR (95% CI)	OR (95%CI)	Poly	Oligo	
Age (years)						
<24 years	1.0(0.4–2.2)	1.5(0.8–3.0)	1.6 (0.8–3.2)	0.8(0.1-4.5)	0.9(0.3–2.3)	
$\geq 24 \text{ years}^{R}$	1	1	1	1	1	
Marital Status						
Unmarried	2.7*(1.1-6.7)	0.7(0.3–1.5)	1.1(0.5–2.4)	2.4(0.3–15.5)	0.9(0.3–2.6)	
Married ^R	1	1	1	1	1	
Occupation						
Working	1.6(0.6–4.5)	0.7(0.3–1.6)	0.6(0.5–2.4)	0.8(0.1–7.1)	0.9(0.3–3.0)	
Non-working	1.6(0.5–4.8)	0.6(0.2–1.6)	1.5 (0.6–3.8)	2.9 (0.4–22.2)	1.0(0.3–3.5)	
Students ^R	1	1	1	1	1	
Diet						
Vegetarian	1.1(0.4–2.8)	2.3*(1.0-5.5)	1.1(0.5–2.7)	0.1(0.01–1.4)	0.1(0.02–1.3)	
Non-vegetarian	0.8(0.3–2.2)	1.9 (0.7–4.6)	1.4(0.6–3.4)	0.1 (0.01–2.0)	0.1(0.02–1.6)	
Ovatarian ^R	1	1	1	1	1	
Frequency of food intake						
Once	0.6(0.05–7.6)	2.9(0.1–2.9)	2.3(0.2–28.1)	1.7 (0.1–1.7)	```	
Twice	0.7(0.3–1.5)	1.1(0.5–2.1)	0.7 (0.3–1.3)	0.1 (0.02–1.7)	0.8(0.3–1.9)	
Thrice ^R	1	1	1	1	1	
Socio-economic status						
High	7.3**(2.3–22.8)	```	3.4**(1.8-10.1)	9.0**(0.7-11.4)	· · · ·	
Upper-middle	1.0 **(0.5-2.1)	$2.9^{**}(1.6-5.4)$	3.1**(1.6-5.7)			
Lower-middle Poor ^R	1.1**(0.1–1.1)	1.8*(1.8–18.4)	1.8* (0.1–1.8)	$1.3^{*}(0.1-1.3)$	1.8(0.1–24.1)	
	1	1	1	1	1	
PAL	11(01 (5)	0.4(0.00, 2.2)	0.0(0.1.4.6)		4 2 (0 7 22 4)	
Sedentary Moderate	1.1(0.1–6.5) 1.4 (0.2–8.5)	0.4(0.09–2.2) 0.7(0.1–3.4)	0.9(0.1–4.6) 0.8 (0.1–4.3)	5.5**(1.3–19.4) 4.6*(0.4–4.6)	$\begin{array}{l} 4.2(0.7-22.4) \\ 4.3(0.8-23.3) \end{array}$	
Vigrous ^R	1.4 (0.2-0.5)	0.7(0.1-3.4)	0.0 (0.1-4.5)	4.0 (0.4-4.0)	4.3(0.8-23.3)	

p<.05*, *p*<.01 **, *p*< .001***.

OR: odds ratios, 95% CI: confidence interval, R Reference category.

CI _1.3–19.4, p < 0.01) more risk for the occurrence of polymenorrhea and had 4.6 times (95% CI _0.4–4.6, p < 0.01) higher risk for the same symptom in subjects with moderately active PAL as compared to vigorously active PCOS women.

Discussion

Polycystic ovary syndrome (PCOS) is a hidden epidemic affecting the female population in their reproductive period and is emerging as one of the most common female endocrinopathies. A study by Gill et al. (2013) estimated 3.7% prevalence of PCOS in North India. In our cross-sectional study out of the total 250 PCOS women, a substantial proportion of women (52.8%) were overweight/ obese, while 43.6% had normal weight, and 3.6% were underweight. It was observed by Frisch (1987) that excessive thinness and obesity have been associated with hypothalamic dysfunction and anovulatory menstrual cycles. Previous research has established that the clinical presentation of PCOS is heterogeneous in nature. In our study the female participants had the following symptom ranges: polycystic ovary (83.2%), hirsutism (74.4%), irregular menstruation (60%), seborrhea (45.2%) and acne (40%).

Category wise frequency distribution of hirsutism was 42% in the overweight/ obese category. A previous study by Majumdar and Singh (2009) also showed that clinical hyperandrogenism among North Indian PCOS women was significantly higher in the obese PCOS group than in lean PCOS women (74.2% vs 50.6%). The study further demonstrated that the prevalence of hirsutism was higher among PCOS women than non-PCOS women (Hartz et al. 1979). Different ethnic populations (Caucasian, Negriod and East Asian) studied by Azziz et al. (2009) mentioned that the prevalence of hirsutism ranged between 65-75%, which was dramatically higher than the expected value. A cross-sectional study by Merkin et al. (2011) reported 23.3% prevalence of hirsutism in four metropolitan areas of the UK. The prevalence of hirsutism in various populations across the globe presents a considerable variability. In Iran it was 42.7% in Yazdc city (Noorbala and Kefaie 2010), 64.9% in Tehran city (Akhyany et al. 2006) and 25.5% in Tehran (Ramezani et al. 2011). It was observed by past studies (Franks, 1989; Kiddy et al., 1990) that obese PCOS women were more likely to express hirsutism and experience menstrual disturbance in comparison to lean women, regardless of testosterone and androgen levels. Recently, Ali and Guidozzi (2020) highlighted health consequences associated with polycystic ovary syndrome and stated that irrespective of their phenotypic manifestation, women with PCOS were metabolically obese. Findings of, Kaur et al. (2020) also recorded dominance of endomorphic component in PCOS women regardless of their BMI category. A study by Balen et al. (1995) mentioned that obesity worsened the presentation of hyperandrogenism in PCOS.

A study performed by Andy et al. (2010) on an English population reported the prevalence of hyperandrogenism, acne and hirsutism (F-G score>6) which were 75.3%, 14.5% and 72.2% respectively in diagnosed PCOS women. Another study indicated that PCOS women had a higher prevalence of hyperandrogenism and acne with polycystic ovarian morphology than women with normal ovarian morphology (Reilly et al. 2014). It has been reported that excessive secretion of androgens in PCOS women leads to skin changes involving hirsutism, acne, seborrhea and androgenetic alopecia (Balen et al. 1995). Our study revealed that the prevalence of oily skin (seborrhea) was 24.4% in overweight/obese PCOS women (p>0.05). A review of previous work reported that the prevalence of seborrhea was 52.5% in PCOS women attending OPD of Care Institute of Medical Sciences, Hyderabad (Gowri et al. 2015), 29% in PCOS women attending Department of dermatology, Srinagar (Abid et al. 2017) and 4% among Indian PCOS patients (Sharma et al. 2008).

A positive family history is known to be a potential risk factor in the development of PCOS. Our study identified that 21.1% PCOS women had a positive family history of PCOS, out of which 11.6% were overweight/obese, 8.8% normal weight and 0.8% who were underweight. Kahsar-miller et al. (2001) noticed a 5 to 6 fold increment in the incidence of PCOS among first-degree female relatives of affected patients in comparison to the general population. Similarly, findings of Legro et al. (1998) reported that 22% sisters of PCOS patients were also affected by PCOS. Similarly, Bharathi et al. (2017) also identified the presence of history of PCOS in 2.5% mothers and 13.6% sisters in urban population of Chennai.

In our study, prevalence of acne was found to be 40%, with the highest frequency noticed among PCOS women in the overweight/obese category (22%) followed by normal weight (16.8%) and underweight women (1.2%). Various researches noted that the prevalence of acne in PCOS women was 67.5% in Hyderabad (Gowri et al. 2015), 48% in Srinagar (Abid et al. 2017) and 64% in the Indian population (Sharma et al., 2008). In PCOS women from Finland, prevalence of acne ranged from 10–34%, which was significantly higher in comparison to their normal counterparts (Voutilainen et al. 2014).

Polycystic ovary morphology is the cardinal feature of PCOS. In our study 83.2% PCOS women reported to have polycystic ovaries (i.e. ovarian volume \geq 10cm³), out of which 44% were in overweight/obese category. Another study conducted on PCOS women of Iran showed 89.1% prevalence of overall polycystic ovarian morphology, out of which 69.2% were in overweight/obese women (Esmaeilzadeh et al. 2015) and likewise, 82.5% in Sri Lanka women (Wijeyaratne et al. 2005). Similarly, a study conducted by Reid et al. (2017) showed that PCOS women with an ovarian volume>10 cm³ exhibited two times more abnormal biochemical marker of insulin resistance than normal ovarian volume women. Insulin is one of the pathophysiological factors leading to PCOS.

In our study the prevalence of oligomenorrhea (>38 days) was 79.6% in PCOS women. Category wise frequency demonstrated that the prevalence of menstrual irregularities was significantly higher in overweight/obese category than the normal weight PCOS women (42.4% vs 34.4%, p>0.05). Similar results were shown by Majumdar and Singh (2009) on North Indian women that the prevalence of menstrual irregularities was significantly higher among obese PCOS women than lean women (79.2% vs 44%). Another cross-sectional study on South Indian PCOS women demonstrated 87.2% irregular menstruation (Joseph et al., 2016). Earlier literature (Kiddy et al. 1990; Liou et al. 2009) has cited that obesity worsens menstrual disturbance in PCOS women. Hence, it is evident from the above discussion that

in the present study symptoms of clinical hyperandrogenism (i.e. hirsutism, seborrhea, oligomenorrhea and excessive as well as moderate father's hair growth) and polycystic ovary were more prevalent in overweight/obese PCOS women, which was further supported by the result of Correspondence Analysis.

In the present study multivariate regression analysis explained that socio-economic status including physical activity, marital status and diet were potential risk factors for the progression of PCOS among females. The odds ratio revealed that the probability to experience hirsutism was 2.7 times more in unmarried women than in married women. Likewise, a study by Merkin et al. (2011) noticed a similar trend with a higher frequency of oligomenorrhea, hirsutism and prevalence of PCOS in younger women. Vegetarian diet had 2.3 folds higher chances to develop seborrhea in reference to ovatarian diet. A study conducted by Bharathi et al. (2017) also noticed that the type of diet is seemed to be the causative and contributing factor towards PCOS disease. Healthy diet and physically active lifestyle contributed towards maintaining normal BMI. The environmental factors such as obesity were also associated with PCOS which can be further exacerbated by poor dietary choices and no physical activity could be a contributing factor in the pathophysiology of PCOS (Diamanti-Kandarakis et al. 2006).

Higher socio-economic status urban women showed higher prevalence of PCOS in India due to sedentary lifestyle and access to high caloric food (Bharathi et al. 2017). Similarly, in our study high class socio-economic status women had the strongest association with clinical symptoms of PCOS exhibiting 7.3 times higher risk to have hirsutism, 8 times likelihood for oily skin occurrence, 4 times more probability to have acne and 9 times more likelihood to experience polymenorrhea followed by upper-middle and lower-middle class. An eco-social framework distinguishes the distribution of disease, as determined via multiple influences comprising biological, environmental, social and historical aspects. This framework depicts results in differentiation in exposures, susceptibilities and resistance to illness (Pathak and Nichter 2015). Previous research observed that high socio-economic status leads to higher prevalence of ovulatory phenotype (Classic PCOS), that seems to be related to adipose tissue type, distribution and insulin levels (Di-Fede et al. 2008).

Findings of a study on urban population documented that females who were not engaged in any physical activity had a higher probability of having PCOS (Bharathi et al. 2017). In accordance to these reports our results also identified that sedentary behavior had a 5.5 times higher risk to develop polymenorrhea, and 4.6 times in moderately active level in reference to women engaged in vigorous physical activity. A study conducted by Carmina et al. (2003) addressed that obesity is the part of disorder of PCOS women, which might have origin in genetic component, dietary factor and lifestyle. Weight reduction has been shown to improve clinical, metabolic and endocrinal factors in PCOS women (Moran et al. 2003).

In the discussion presented, it is evident that obesity represents as a significant risk factor, which can increase many of the symptoms of PCOS. With increase in weight both oestrogen and androgen production increases (Gambineri et al. 2002) which is one of the causative factors responsible for menstrual disturbance.

The following are the limitations of the present study:

– few participants were not open and comfortable to answer especially the questions related to their reproductive health. The data collected concerning reproductive variables, socio-demographic characteristics and family histories were self-reported by the participants. Hence, there was a probability of biased reporting. Women were asked about their age at menarche, which may have also increased the risk of biased reporting.

Conclusion

PCOS is one of the most prevalent non-communicable hormonal disorders worldwide with unknown pathophysiology. Obesity prevalence is a common finding in PCOS women affecting reproductive and metabolic features. Our study also revealed that women in the overweight/obese category had a higher prevalence of various symptoms associated with PCOS. Our study confirmed that Obesity as being a potential factor in increasing symptoms associated with PCOS. Multivariate regression model assessed sedentary lifestyle, high socio-economic status, vegetarian diet and unmarried women as possible determinants for the development of PCOS. Furthermore, all these factors leading to overweight/obese body build which are again act as contributing factor to worsen the symptoms of PCOS. Consequently, weight management may be beneficial in controlling PCOS symptoms. Therefore, study emphasizes the need for more scientific attention in this area.

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The Authors' contributions

MK, RK and VS are involved in study conception and research design and manuscript drafting. RK involved in collection of data. RK and MK analyzed and interpreted the data.

Conflict of interest

The authors declare that there is no conflict of interest.

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References

- Abid Keen M, Hassan SI, Gousia S. 2017. Cutaneous manifestations of polycystic ovary syndrome: A cross-sectional clinical study. Indian Dermatol Online J 8(2):104– 10. doi: 10.4103/2229-5178.202275.
- Agarwal OP, Bhasin SK, Sharma AK, Chabra P, Agarwal K, Rajoura OP. 2005. A new instrument (scale) for developing socio-

economic status of a family. Preliminary Study. Indian J Community Med 30:111– 4.

- Ahmadi A, Akbarzadeh M, Mohammadi F, Akbari M, Jafari B. 2017. Anthropometric characteristics and dietary pattern of women with polycystic ovary syndrome. Indian J of Endo and Metabol :17(4):672– 6. doi: 10.4103/2230-8210.113759.
- Akhyany M, Daneshpajouh M, Barzegari M, Ghandi N, Ghiasi M, Chenari Z, et al. 2006. The prevalence of hirsutism in female students of Tehran University School of Medicine. J Dermatol 9:242–9.
- Ali AT, Guidozzi F. 2020. Midlife women health consequences associated with polycystic ovary syndrome. Climactric 23(2):116–22. doi: 10.1080/13697137.2019.1679111.
- Ali M, Cleland J. 1999. Determinants of contraceptive discontinuation in six developing countries. J Biosoc Sci 31(3):343–60.
- Andy H, Brennan K, Azziz R. 2010. Prevalence of hyperandrogenemia in the polycystic ovary syndrome diagnosed by the National Institutes of Health 1990 criteria. Fertil Steril 93(6):1938–41. doi: 10.1016/j.fertnstert.2008.12.138.
- Anjana RM, Sudha V, Lakshmipriya N, Subhashini S, Pradeepa R, Geetha L. et al. 2015. Reliability and validity of a new physical activity questionnaire for India. Int J Behav Nutr Phys Act 18:12–40. doi: 10.1186/s12966-015-0196-2.
- Azziz R, Carmina E, Chen Z, Dunaif A, Laven JS, Leg RS. et al. 2016. Polycystic ovary syndrome. Nat Rev Dis Primers 11(2):16057. doi: 10.1038/nrdp.2016.57.
- Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W. et al. 2009. The androgen excess and PCOS society criteria for the polycystic ovary syndrome: the complete task force report. Fertil Steril 91:456–88. doi: 10.1016/j.fertnstert.2008.06.035.
- Sam S, Dunaif A. 2003. Polycystic ovary syndrome: Syndrome XX?. Trends in Endocrinol Metab 14(8):365–70. doi:10.1016/j. tem.2003.08.002.

- Balen AH, Conway GS, Kaltsas G, Techatrasak K, Manning PJ, West C. et al. 1995. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod 10(8):2107–11.
- Balen AH, Laven JS, Tan SL, Dewailly D. 2003.Ultrasound assessment of the polycystic ovary: international consensus definitions. Hum Reprod Update 6:505–14. doi:10.1093/humupd/dmg044.
- Bernasconi D, Del Monte P, Meozzi M, Randazzo M, Marugo A, Badaracco B. et al. 1996. The impact of obesity on hormonal parameters in hirsute and nonhirsute women. Metabolism 45(1):72–5. doi:10.1016/ s0026-0495(96)90202-4.
- Bharathi RV, Swetha S, Neerajaa J, Madhavica V, Janani DM, Rekha SN, et al. 2017. An epidemiological survey: Effect of predisposing factors for PCOS in Indian urban and rural population. Middle East Fertil Soc 22(4):313–6. doi:10.1016 /j. mefs. 2017.05.007.
- Carmina E, Legro RS, Stamets K, Lowell J, Lobo RA. 2003. Difference in body weight between American and Italian women with polycystic ovary syndrome: influence of the diet. Human Reproduction 18(11):2289–93.
- Charan J, Biswas T. 2013. How to calculate sample size for different study designs in medical research?. Indian J Psychol Med 35(2):121–6. doi:10.4103/0253-7176.116232.
- Diamanti-Kandarakis E, Kandarakis H, Legro RS. 2006. The role of genes and environment in the etiology of PCOS. Endocrine 1:19–26. doi:10.1385/ENDO:30:1:19.
- Diamanti-Kandarakis E, Papavassiliou AG, Kandarakis SA, Chrousos GP (2007. Pathophysiology and types of dyslipidemia in PCOS. Trends Endocrinol Metab 18(7):280–5. doi: 10.1016/j. tem.2007.07.004.
- Di-Fede G, Mansueto P, Longo R, Rini GB, Carmina E. 2008. Influence of sociocultural factors on the ovulatory status of polycystic ovary syndrome. Fertil Ster-

il 91(5):1853–6. doi: 10.1016/j.fertnstert.2008.02.161.

- Esmaeilzadeh S, Andarieh MG, Ghadimi R, Delavar MA. 2015. Body Mass Index and Gonadotropin Hormones (LH & FSH) Associate With Clinical Symptoms Among Women With Polycystic Ovary Syndrome. Glob J Health Sci.7(2):101–6. oi: 10.5539/ gjhs.v7n2p101.
- Ekwutosi MO, Hooper WC, Atrash HK, Yusuf HR, Boulet SL. 2012. Prevalence of polycystic ovary syndrome among privately insured, United States, 2003–2008. Am J Obstet Gynecol 207(4):299.e1-299.e7. doi: 10.1016/j.ajog.2012.07.023.
- Franks S. 1989. Polycystic ovary syndrome: a changing perspective. Clin Endo 31:87–120. doi.org/10.1111/j.1365-2265.1989. tb00457.x.
- Franks S, Kiddy D, Sharp P, Singh A, Reed M, Seppälä M. et al. 1991. Obesity and polycystic ovary syndrome. Ann N Y Acad Sci 626:201–6. doi: 10.1111/j.1749-6632.1991.tb37915.x.
- Frisch RE. 1987. Body fat, menarche, fitness and fertility. Hum Reprod 2(6):521–33.
- Gambineri A, Pelusi C, Vicennati V, Pagotto U, Pasquali R. 2002. Obesity and the polycystic ovary syndrome. Int J Obes Relat Metab Disord 26:883–96. doi:10.1038/sj.ijo.0801994.
- Gill H, Tiwari P, Dabadghao P. 2013. Prevalence of polycystic ovary syndrome in young women from North India: A Community-based study [published correction appears in Indian]. J Endocrinol Metab 17(1):162. doi:10.4103/2230-8210.10410.
- Gowri BV, Chandravathi PL, Sindhu PS, Naidu KS. 2015.Correlation of skin changes with hormonal changes in polycystic ovarian syndrome: A cross sectional study clinical study. Indian journal of dermatology. 60(4):419. doi:10.4103/0019-5154.160505.
- Hartz AJ, Barboriak PN, Wong A, Katayama KP, Rimm AA. 1979. The association of obesity with infertility and related men-

strual abnormalities in women. Int J Obes 3(1):57–77.

- Holte J, Gennarelli G, Wide L. 1994. The independent effects of polycystic ovary syndrome and obesity on serum concentrations of gonadotropins and sex steroids in premenopausal women. Clin Endocrinol (Oxf) 41:473–81.
- Hsu MI. 2015. Clinical characteristics in Taiwanese women with polycystic ovary syndrome. Clin Exp Reprod Med 42(3):86– 93. doi:10.5653/cerm.2015.42.3.86.
- Joseph N, Reddy AG, Joy D, Patel V, Santhosh P, Das S. et al. 2016. Study on the proportion and determinants of polycystic ovarian syndrome among health sciences students in South India.J Nat SciBiol Med 7(2):166–72. doi:10.4103/0976-9668.184704.
- Kahsar-Miller MD, Christa Nixon BS, Boots LR, Go RC, Azziz R. 2001. Prevalence of polycystic ovary syndrome (PCOS) in first-degree relatives of patients with PCOS. Fertil Sertil 75(1):53–8. doi: 10.1016/s0015-0282(00)01662-9.
- Kauffman RP, Baker VM, Dimarino P, Gimpel T, Castracane VD. 2002. Polycystic ovarian syndrome and insulin resistance in white and Mexican American women: A comparison of two distinct population. Am J Obstet Gynecol 187(5):1362–9. doi:10.4239/wjd.v2.i3.33.
- Kaur R, Kaur M, Suri V. 2020. Somatotype profile of obese and lean women with polycystic ovary syndrome: a population based cohort study. Anthropologie (Brno) 58(1):93–102. doi: https://doi. org/10.26720/anthro.20.02.07.1.
- Kiddy DS, Sharp PS, White DM, Scanlon MF, Mason HD, Bray CS. et al. 1990. Differences in clinical and endocrine features between obese and non obese subjects with polycystic ovary syndrome: an analysis of 263 consecutive cases. Clin Endocrinol 32(2):213–20.
- Legro RS, Driscoll D, Strauss JF, Fox J, Dunaif A. 1998. Evidence for a genetic basis for hyperandrogenemia in polycystic ovary syndrome. PNAS 8, 95(25):14956–60.

- Liou TH, Yang JH, Hsieh CH, Lee CY, Hsu CS, Hsu MI. 2009. Clinical and biochemical presentations of polycystic ovary syndrome among obese and nonobese women. Fertil Steril 92(6):1960–5. doi:10.1016/j.fertnstert.2008.09.003.
- Majumdar A, Singh TA. 2009. Comparison of clinical features and health manifestations in lean vs. obese Indian women with polycystic ovarian syndrome. J Hum Reprod Sci 2(1):12–17. doi: 10.4103/0974-1208.51336.
- Merkin SS, Azziz R, Seeman T, Calderon-Margalit R, Daviglus M, Kiefe C. et al. 2011 Socio-economic status and polycystic ovary syndrome. J Womens Health (Larchmt) 20(3): 413-9. doi: 10.1089/ jwh.2010.2303.
- Moran LJ, Noakes M, Clifton PM, Tomlinson L, Norman RJ. 2003. Dietary Composition in Restoring Reproductive and Metabolic Physiology in Overweight Women with Polycystic Ovary Syndrome. J Clin Endocrinol Metabol 88(2):812–9. doi:10.1210/ jc.2002-020815.
- Munro MG, Critchley HOD, Fraser IS. 2018. The two FIGO systems for normal and abnormal uterine bleeding symptoms and classification of causes of abnormal uterine bleeding in the reproductive years: 2018 revisions. Int J Gynecol Obstet 143:393–408. doi: 10.1002/ijgo.12666.
- Noorbala MT, Kefaie P. 2010. The Prevalence of Hirsutism in Adolescent Girls in Yazd, Central Iran. Iran Red Crescent Med J 2:111–117.
- Pathak G, Nichter M. 2015. Polycystic ovary syndrome in globalizing India: an ecosocial perspective on an emerging lifestyle disease. Soc Sci 146:21–8. doi:10.1016/j. socscimed.2015.10.007.
- Ramezani TF, Simbar M, Tohidi M, Hosseinpanah F, Azizi F. 2011. The prevalence of polycystic ovary syndrome in a community sample of Iranian population: Iranian PCOS prevalence study. Reprod Biol Endocrinol 39:1–7. doi: 10.1186/1477-7827-9-39.

- Reid SP, Chia-Ning K, Pasch L, Shinkai K, Cedars M, Huddleston H. et al. 2017. Ovarian morphology is associated with insulin resistance in women with polycystic ovary syndrome: a cross sectional study. Fertility Research and Practice 3:1–7. doi:10.1186/ s40738-017-0035-z.
- Reilly MW, Taylor AE, Crabtree NJ, Hughes BA, Capper F, Crowley RK. et al. 2014. Hyperandrogenism predicts metabolic phenotype in polycystic ovary syndrome: the utility of serum androstenedione. J Clin Endocrinol Metab 99(3):1027–36. doi: 10.1210/jc.2013-3399.
- Rotterdam ESHŔE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 (2004) consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 81(1):19–25. doi:10.1016/j.fertnstert.2003.10.004.
- Sharma, N.L., Mahajan, V.K., Jindal, R., Gupta, M., Lath, A. (2008).Hirsutism: Clinico-investigate profile of 50 Indian patients. Indian J Dermatol. 53(3), 111–114. Doi: 10.4103/0019-5154.42387
- Tamimi W, Siddiqui I, Tamim H, Al Eisa N, Adham M. 2009. Effect of body mass index on clinical manifestations in patients with polycystic ovary syndrome. Int J Gynecol Obstet 107(1):54–7. doi: 10.1016/j. ijgo.2009.06.003.
- Tanner JM, Whitehouse RH, Takaishi M. 1966. Standards from birth to maturity for height, weight, height velocity and weight velocity in British children. Archieve Dis Child 41:454–613.
- Voutilainen R, Jaaskelainen J. 2014. Premature adrenarche: Etiology, clinical findings, and consequences. J Steroid Biochem Mol Biol 145:226–36. doi: https:// doi.org/ 10.1016 /j.jsbmb.2014.06.004.
- Welt CK, Gudmundsson JA, Arason G, Adams J, Palsdottir H, Gudlaugsdottir G. et al. 2006.Characterizing discrete subsets of polycystic ovary syndrome as defined by the Rotterdam criteria: the impact of weight on phenotype and metabolic features. J Clin Endocrinol

Metab 91(12):4842–8. doi: https://doi. org/10.1210/jc.2006-1327.

- WHO expert consultation. 2004. Appropriate body-mass index for Asian population and its implications for policy and intervention strategies. Lancet 363:157–63. doi: 10.1016/S0140-6736(03)15268-3
- Wijeyaratne CN, Jayasinghe A, de Silva DG, Parkes AB, Lazarus JH, Premawardhana LD. 2005. Iodine prophylaxis, goitre and thyroid autoimmunity in Sri Lanka. Ceylon Med J 50(1):20–23. doi: 10.4038/cmj. v50i1.1585.