Original Article

Clinical, biochemical, and radiological profile of polycystic ovary syndrome in adolescents attending an obesity clinic

Annapoorna Muthukumar[1], Varun Govind Krishna[2], Hemchand Krishna Prasad[3], Kannan Narayanaswamy[4], C Vasudevan Ravisekar[4], Nedunchelian Krishnamoorthy[4]

From [1]Pediatrician, Department of Paediatrics, [2]Research Assistant, Department of Pediatric Endocrinology, [3]Consultant, Department of Paediatric Endocrinology and Diabetes, [4]Consultant, Department of Paediatrics, Mehta Multispeciality India Private Limited, Chennai, Tamil Nadu, India

ABSTRACT

Background: Paucity of data on adolescent polycystic ovary syndrome (PCOS) from South India. **Objective:** The objective of the study was to describe the clinical and biochemical profile of PCOS and its relationship to cardiometabolic risk in obese adolescent girls. **Materials and Methods:** A prospective observational study was conducted on adolescent girls attending obesity clinic over a 3-year period. Overweight and obese adolescent girls diagnosed as PCOS (based on Rotterdam criteria) were considered as Group I and without PCOS considered as Group II. A detailed history, physical examination, markers of hyperandrogenism, and metabolic profile were assessed and compared in two groups. Prevalence of metabolic syndrome and individual cardiometabolic risk factors was ascertained. All subjects were initiated on lifestyle measures and medications as clinically indicated. **Results:** Thirty-three girls with PCOS (Group I) and 33 without PCOS (Group II) were recruited. About 72.7% of subjects showing oligo or anovulatory cycles, 51.5% showed biochemical hyperandrogenism (mean testosterone level 36.76 ± 9.86 ng/dL), 69.7% showed clinical hyperandrogenism, and 84.5% showed radiological evidence of PCOS. The percentages of subjects fulfilling two and all three criteria of Rotterdam criteria for PCOS were 75.75% and 24.24%, respectively. Total 36% of the subjects had positive family history. Hirsutism was present in 54.54% (mean Ferriman-Gallwey score 8.6 ± 2.17). Percentage of subjects with individual metabolic abnormalities increased across the tertiles of BMI irrespective of the presence of PCOS. **Conclusion:** The profile of PCOS in obese South Indian adolescent girls is described. All overweight and obese adolescent girls should be screened for metabolic abnormalities, irrespective of the presence of PCOS.

Key words: Cardiometabolic risk, Hyperandrogenism, Polycystic ovary syndrome

Childhood and adolescent obesity is on the rise in our country [1,2]. Urbanization, unhealthy diets, and increasingly sedentary lifestyles have contributed to increase in the prevalence of childhood obesity; particularly in developing countries, obesity is an important problem in young adolescent girls [3]. There is an increase in sedentary lifestyle and increase in social and emotional expectations from an adolescent girl to concentrate on scholastic performance rather than physical activity. Polycystic ovary syndrome (PCOS) is a syndrome of acne, weight gain, menstrual irregularity, and insulin resistance seen in obese adolescent girls, often seen by the pediatricians [4]. This condition is characterized by chronic anovulation, hyperandrogenism, and polycystic ovaries and exhibits marked heterogeneity in its clinical manifestations. Most adolescents with PCOS have metabolic abnormalities such as insulin resistance with compensatory hyperinsulinemia, obesity, and dyslipidemia from menarche [5]. All these metabolic features may play a role in the development of glucose intolerance or type 2 diabetes mellitus and hypertension; thereby, increasing the risk of developing cardiovascular diseases [6].

The previous researches on PCOS in our nation have been performed from different centers in Asian countries [7,8]. Adolescent PCOS has been studied in various parts of India [9-13]. These studies describe PCOS in adolescent girls at a community level, none from a specialized obesity clinic. A timely diagnosis of PCOS is pivotal for the initiation of an appropriate therapy [14]. There is a need to study the profile and determine the characteristics of the condition to rationalize management of this condition, which may have long-term health implications.

Risk factor for development of metabolic syndrome in obese adolescents can be either nutritional driven or related to

Access this article online

Received - 22 November 2020
Initial Review - 13 December 2020
Accepted - 18 January 2021

DOI: 10.32677/IJCH.2021.v08.i01.004

Quick Response code

Correspondence to: Hemchand Krishna Prasad, Department of Paediatric Endocrinology and Diabetes, Mehta Multispeciality Hospitals India Pvt. Ltd., Chennai, Tamil Nadu, India. E-mail: pediatricendocrinology.mehta@gmail.com

endocrinological abnormality (PCOS). The role of PCOS as an additive factor contributing to additional metabolic risk in obese adolescent girls is debatable [15-18]. Hence, we performed this study to describe the clinical, biochemical, and radiological profile of South Indian adolescent girls with PCOS and also describe its role in development of metabolic abnormalities in adolescent girls attending obesity clinic (Fig. 1).

METHODS

We conducted a prospective observational study in a tertiary care hospital in South India, over a period of 3 years with approval from Institutional Scientific and Institutional Ethics Committees. Adolescent girls attending obesity clinic in the hospital were the study population. All adolescent girls 2 years after menarche presenting to obesity clinic with BMI >23 adult equivalent as per the new growth charts of the Indian academy of pediatrics [19], who consented for the study were included in the study. Syndromic obesity, drug induced obesity, and obesity secondary to intracranial space occupying lesions, patients who did not consent for the study, were excluded from the study.

Adolescent girls were diagnosed as PCOS as per the conventional Rotterdam criteria [20] as shown in Table 1. A detailed history (menstrual irregularity, features of hyperandrogenism, galactorrhea, and family history of PCOS); clinical feature assessment (features of hyperandrogenism, hirsutism, and blood pressure); anthropometric measurements (height, weight, and body mass index (BMI); and waist circumference were performed in all subjects. Ultrasound abdomen was performed by a single blinded radiologist to look for ovarian morphology. Hirsutism was assessed by the Ferriman-Gallwey score [21]. Waist circumference and blood pressure measurements were converted into Z-sores using appropriate references [22]. Overweight and obese adolescent girls diagnosed as PCOS were considered as Group I and without PCOS was considered as Group II.

Blood investigations such as lipid profile, blood sugar in fasting and postprandial state, serum insulin in fasting, and postprandial state, sex hormones including LH, FSH, and total testosterone were done in the follicular phase (day 5 after cessation of menses). Subjects were divided into two groups: Those with and without PCOS (defined in Table 1). Other investigations such as thyroid functions including free T4 and TSH, prolactin, overnight dexamethasone suppression test for Cushing's syndrome, and synacthen stimulation test (for non-classical congenital adrenal hyperplasia) were individualized for specific subjects according to the clinical presentation. The prevalence of metabolic syndrome and individual cardiometabolic risk factors (Table 1) was compared in adolescent girls with and without PCOS.

All the subjects with and without PCOS were initiated on lifestyle measures including diet and exercise. Subjects with PCOS were treated with oral contraceptive pills; metformin or anti-androgens are per the clinical situation. Girls with severe hirsutism were referred to the dermatologist for cosmetic laser therapy after hormonal control.

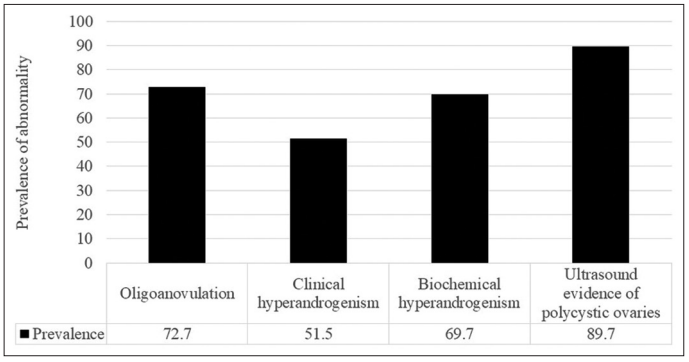

Figure 1: Percentage of study subjects satisfying individual Rotterdam criteria

Statistical Analysis

Based on an Indian study [23], the standard deviation of BMI score is 3.07. Required sample size was 27 women per group to demonstrate 2.5 BMI mean difference between study and control group (25.0–22.5) with a power of the study 85% (type II error $\beta = 15\%$) and at a 95% confidence of result (type 1 error $\alpha = 5\%$). After allowing 15% of dropout rate, final sample size was 66 girls (33 study + 33 control). Data entry was done using predesigned data entry sheet. Data were imported to SPSS and analyzed using

Table 1: Study definitions

Study parameter	Definition
Metabolic syndrome (23)	Metabolic syndrome is present if three or more of the following five criteria are met: a) Waist circumference >40 inches (men) or 35 inches (women), b) Blood pressure >130/85 mmHg, c) Fasting TG level >150 mg/dL, d) Fasting HDL cholesterol <50 mg/dL (women) e) Fasting blood sugar >100 mg/dL.
PCOS	Rotterdam criteria (20) Two of the three criteria a) Oligo or anovulation b) Polycystic ovary on ultrasound (12 or more cysts in single ovary or ovarian volume more than 10 cubic.mm) c) Clinical and/or biochemical hyperandrogenism
Amenorrhoea (8)	Absence of periods for a length of time equivalent to a total of at least 3 of the previous cycle intervals or 6 months of amenorrhea.
Oligomenorrhea. (14)	Menstrual intervals persistently shorter than 20 days or >45 days in girls >2 years after onset of menarche
Overweight (19)	BMI >23 kg/m^2 adult equivalent
Obesity (19)	BMI >27 kg/m^2 adult equivalent
HOMA-IR	$\frac{\text{Fasting glucose} \times \text{Fasting insulin}}{405}$

PCOS: Polycystic ovary syndrome, HDL: High-density lipoprotein, TG: Triglyceride, BMI: Body mass index, HOMA-IR: Homeostatic model for assessment of insulin resistance

SPSS 21.0. Descriptive statistics were used to analyze the baseline variables. Intergroup and intragroup comparisons of categorical variables were done by Chi-square test and one-way ANOVA test as applicable. $P < 0.05$ is considered for statistical significance.

RESULTS

Out of all the obese adolescents who attended our obesity clinic, 66 were recruited to our study, 33 had PCOS satisfying the Rotterdam criteria (Group I), and other 33 were without PCOS (Group II). The percentage of subjects satisfying each criterion described by Rotterdam criteria included: About 72.7% showed oligo or an ovulatory cycles, 51.5% showed biochemical hyperandrogenism, 69.7% showed clinical features of hyperandrogenism, and 84.5% of the subjects showed radiological evidence of PCOS. Total 75.75% and 24.24% subjects fulfilled two and all three criteria of Rotterdam criteria for PCOS, respectively.

The profile of PCOS is given in Table 2. The presence of PCOS was found to be 50% in young adolescent overweight and obese girls in our study. Four out of 33 (12.12%) subjects with PCOS had hypothyroidism, 5 out of 33 (Group I) (15.15%) had marginal elevation in prolactin levels – not warranting therapy. All

Table 2: Profile of girls with PCOS

Study parameter	Group I with PCOS (*n*=33)	Group II without PCOS (*n*=33)
Clinical features		
Age (in years)	15.5±2.4	12.5±1.8
Positive Family history in subjects with PCOS[a]	12 (36%)	4 (12.1%)
BMI (kg/m^2)	27.4±4.8	26.2±43
Waist circumference mean Z score	1.2±0.9	1.1±08
Systolic BP mean Z score	0.7±1.2	0.6±1.0
Diastolic BP mean Z score	0.8+0.9	0.7±0.9
Mean Ferrimen-Gallwey score	8.6±2.1	6.1±1.8
Hirsuitism	18 (54.5%)	2 (6.0%)
Temporal balding	10 (30.3%)	0 (0%)
Acne	13 (39.3%)	0 (0%)
Acanthosis	15 (45.4%)	18 (54.5%)
Clitoromegaly	1 (3.03%)	0 (0%)
Voice change	2 (6.06%)	0 (0%)
Skin tags	2 (6.06%)	0 (0%)
Biochemical parameters		
LH (mIU/mL)	7.7±4.3	5.9±1.5
FSH (mIU/mL)	5.1±1.7	5.0±1.3
Testosterone (ng/dL) (normal <50)[a]	36.7±9.8 (54.1%)	29.7±10.3 (3.0%)
Prolactin (ng/mL) (normal <25)	20.0±15.0 (15.15%)	10.3±5.5 (15.1%)
LF/FSH ratio (normal 1)[a]	2.2±3.5 (31.8%)	1.1±0.6 (12.1%)
Metabolic parameters		
TGL (mg/dL)	89.9±42.3 (30.4%)	109.5±23.4 (18.1%)
LDL (mg/dL)	93.2±21.0 (37.5%)	94.0±19.5 (33%)
HDL (mg/dL)	40.2±7.1 (43.4%)	38.2±10.2 (30.3%)
Fasting sugar (mg/dL)	86.2±11.5 (6%)	90.5±12.8 (12.1%)
Postprandial sugar (mg/dL)	116.6±30.5 (6%)	122.2±32.9 (18.1%)
Fasting insulin (mIU/L)	29.9±19.2 (55%)	28.8±10.6 (33.3%)
Postprandial insulin (mIU/L)	311.3±30.6 (60%)	155.0±19.4 (63.6%)
Radiological parameters		
Bilateral polycystic ovaries in USG	84.8%	0%
Treatment		
Lifestyle modifications	33 (100%)	33 (100%)
Pharmacotherapy		
Metformin	13 (39.4%)	4 (12.1%)
Oral contraceptive agents	4 (12.1%)	0 (0%)
Anti androgens	0 (0%)	0 (0%)
Cosmetic laser therapy	1 (3%)	0 (0%)

[a]**p<0.05**

the subjects had appropriate cortisol suppression with overnight dexamethasone suppression test. In three out of 33 subjects, non-classical congenital adrenal hyperplasia was considered, for which a synacthen stimulation test was performed. The cortisol response to synacthen in all the three cases was adequate (>20 mcg/dL) with normal 17-hydroxy progesterone response.

About 36% of the subjects with PCOS had a positive family history of PCOS. Hirsutism was the most predominant clinical hyperandrogenic marker, and it was present in 54.54% of the PCOS cases with a mean Ferriman-Gallwey score of 8.6 ± 2.17. The mean testosterone level was 36.76 ± 9.86 ng/dL (54.16% of the subjects had abnormal testosterone levels). Clinical acanthosis and biochemical hyperinsulinemia was present in 45% and 55% of the subjects, respectively. The mean LH/FSH ratio was 2.28 ± 3.56 in the follicular phase (31.8% of the subjects with PCOS had an abnormal LH/FSH ratio), parallelly 72.7% of the study subjects had oligo or anovulation (Table 2).

To further elucidate the relationship between PCOS and cardiometabolic risk, we compared the percentage of overweight and obese adolescent girls with and without PCOS who had specific metabolic abnormalities (Fig. 2a). The cardiometabolic risk factors (abnormal HOMA, LDL, HDL, fasting insulin, and fasting sugar) were comparable in the two groups with no significant difference ($p > 0.05$) (Fig. 2b). We constructed tertiles of BMI in the study subjects with and without PCOS. It was observed that the percentage of subjects with individual metabolic abnormalities increased across the tertiles of BMI (Fig. 2c). This was not impacted by the presence or absence of PCOS.

All the study subjects were advised on diet, exercise, and lifestyle measures. Subjects in Group I were initiated on metformin, oral contraceptives and anti-androgens as per the clinical indication (Table 2). Cosmetic laser therapy was recommended after control of hormonal status in one subject.

DISCUSSION

To the best of our knowledge, this is the first study from our part of the country describing adolescent PCOD. We have described the profile of obese adolescent girls with PCOS evaluated and being managed in the obesity clinic of a multi-disciplinary pediatric hospital. In our series, we observed that our subjects have higher insulin resistance, hyperandrogenism, abnormal ovarian morphology, and significant familial clustering compared to other series. We also noted that in obese adolescent girls, presence of PCOS did not add to the risk of cardio-metabolic complications.

It is observed that our subjects were younger (mean age 15.5 years) compared to the previous published reports, because we have recruited only adolescents for our study. Our girls had a higher BMI (27.4 kg/m^2) compared to other series as our sample is recruited in the obesity clinic, whereas other series have recruited their population in colleges, universities [11-13] or in the community [7,8]. We had 72.7% of our girls with oligoanovulation. This is probably due to our strict definition of oligomenorrhea of 45 days (14) versus the conventional 35 days [7,8] cutoffs used in other series. It was also observed that 36% of our subjects had a first degree relative with PCOS in line with other studies. This has been seen in western studies as

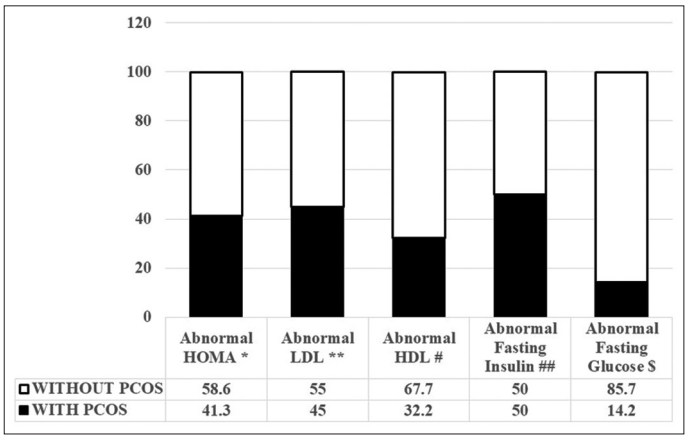

Figure 2a: Comparison of abnormal metabolic parameters among subjects with and without PCOS. *HOMA > 2.5, **LDL > 100 mg/dL, #HDL < 40 mg/dL, ##Fasting insulin > 25 mIU/L, $fasting glucose > 100 mg/dl. None p<0.05

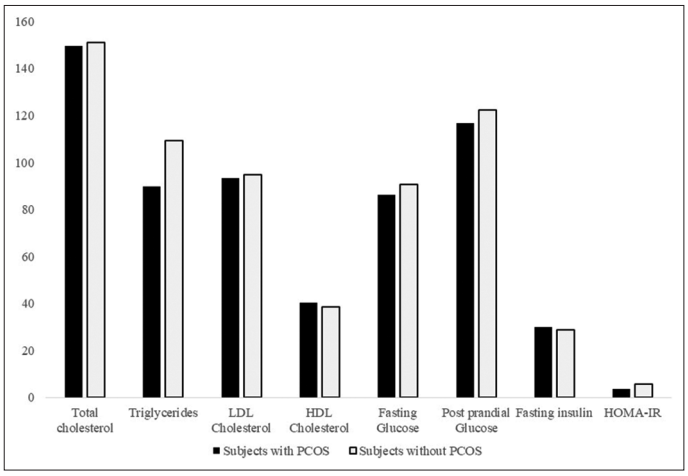

Figure 2b: Comparison of mean metabolic parameters among subjects with and without PCOS. Total cholesterol, TGL, LDL, HDL, Glucose depicted as mg/dL; insulin depicted as mIU/mL and HOMA-IR as absolute values. None p<0.05

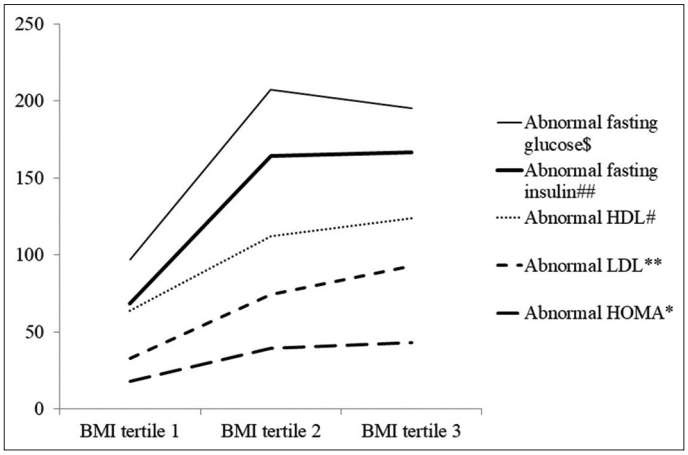

Figure 2c: Distribution of abnormal metabolic parameters among BMI tertiles. *HOMA > 2.5, ** LDL > 100 mg/dL, #HDL < 40 mg/dL, ##Fasting insulin > 25 mIU/L, $Fasting glucose >100 mg/dL

well [24,25]; suggesting a polygenic inheritance of the disease, similar to type 2 diabetes.

We observed that the mean metabolic abnormalities and percentage of individuals with metabolic abnormalities are similar in adolescents with and without PCOS. Higher percentage of metabolic abnormalities was noted across tertiles, irrespective of presence of PCOS. In contrast to our observation – a study done by Coviello *et al*. [15] concluded that adolescent girls with PCOS have a higher prevalence of metabolic syndrome than the general adolescent population and hyperandrogenemia is a risk factor for metabolic syndrome independent of obesity and insulin resistance. In agreement with our results, a cross-sectional study conducted by Rossi *et al*. [17] measured the prevalence of metabolic syndrome and its components in adolescent subjects and controls which states that obese adolescent women had a high prevalence of metabolic syndrome, and PCOS did not add additional risk for metabolic syndrome. On similar lines, a study by Barber *et al*. [18] comparing two groups of BMI matched adolescents with and without PCOS observed that the fat distribution in the two groups was very much similar. Thus, one could conclude that the nutritional component of obesity supersedes the endocrine component in PCOS and predisposes to the development of metabolic complications.

We wish to clarify certain aspects of the study. Our study illustrates the profile of PCOS in obese adolescent girls; we have not included girls with PCOS and normal BMI. Puberty is a phase of physiological hyperinsulinemia and hyperandrogenism which settles as girls reach stage 5 Tanner [26]. To avoid overdiagnosis of PCOS due to these transitory changes, we have enrolled only girls 2 years post-menarche. The diagnostic criterion of PCOS, in adolescence, is a matter of controversy [27]. The diagnosis may be made by the NIH criteria, Rotterdam criteria [20], and the Androgen Excess Society criteria [28]. We have adopted the Rotterdam criteria by convention, as it is the practice in many pediatric endocrine units of our country.

Limitation of our study was that the body fat percentage by DEXA scan was not measured among the subjects. However, we measured waist circumference Z scores as a surrogate marker for body fat percentage. To strengthen our findings, we need a comparison group of non-obese subjects but we could not perform the evaluation of non-obese subjects due to ethical considerations. The limitation of cross-sectional data on cardiometabolic risk prediction is well known. However, these subjects are under follow-up in our endocrinology clinic and will be reassessed periodically.

CONCLUSION

The profile of PCOS in obese South Indian adolescent girls is described in our study. It is recommended that pediatricians have a high index of suspicion for PCOS in all overweight and obese adolescent girls and screen them for metabolic abnormalities, irrespective of the presence of PCOS.

REFERENCES

1. Raja MW, Nazir A, Haq I, Ahmad MM. Childhood overweight and obesity in an urban population of Kashmir-a cross-sectional study. Int J Med Sci Public Health 2016;5:1404-7.
2. Patnaik L, Pattanaik S, Sahu T, Rao EV. Overweight and obesity among adolescents, a comparative study between government and private schools. Indian Pediatr 2015;52:779-781.
3. Juonala M, Magnussen CG, Berenson GS, Venn A, Burns TL, Sabin MA, *et al*. Childhood adiposity, adult adiposity, and cardiovascular risk factors. N Engl J Med 2011;365:1876-85.
4. Kamboj MK, Bonny AE, Polycystic ovary syndrome in adolescence: Diagnostic and therapeutic strategies. Transl Pediatr 2017;6:248-55.
5. Rosenfield RL, Ehrmann DA. The pathogenesis of polycystic ovary syndrome (PCOS). Endocr Rev 2016;37:467-520.
6. Azziz R, Marin C, Hoq L, Badamgarav E, Song P. Health care-related economic burden of the polycystic ovary syndrome during the reproductive life span. J Clin Endocrinol Metab 2005;90:4650-8.
7. Li L, Chen X, He Z, Zhao X, Huang L, Yang D. Clinical and metabolic features of polycystic ovary syndrome among Chinese adolescents. J Pediatr Adolesc Gynecol 2012;25:390-5.
8. Ma YM, Li R, Qiao J, Zhang XW, Wang SY, Zhang QF, *et al*. Characteristics of abnormal menstrual cycles and polycystic ovary syndrome in community and hospital population. Chin Med J 2010;123:2185-9.
9. Gill H, Tiwari P, Dabadghao P. Prevalence of polycystic ovary syndrome in young women from North India: A community-based study. Indian J Endocrinol Metab 2012;16:S389.
10. Joshi B, Mukherjee S, Patil A, Purandare A, Chauhan S, Vaidya R. A cross-sectional study of polycystic ovarian syndrome among adolescent and young girls in Mumbai, India. Indian J Endocrinol Metab 2014;18:317.
11. Nidhi R, Padmalatha V, Nagarathna R, Amritanshu R. Prevalence of polycystic ovarian syndrome in Indian adolescents. J Pediatr Adolesc Gynecol 2011;24:223-7.
12. Vijayan CP, Sonia A. Prevalence of polycystic ovary syndrome among students of a teaching collegiate hospital. Health Sci 2013;2:1-11.
13. Joseph N, Reddy AG, Joy D, Patel V, Santhosh P, Das S, *et al*. Study on the proportion and determinants of polycystic ovarian syndrome among health sciences students in South India. J Nat Sci Biol Med 2016;7:166-72.
14. Witchel SF, Oberfield S, Rosenfield RL, Codner E, Bonny A, Ibanez L, *et al*. The diagnosis of polycystic ovary syndrome during adolescence. Hormone Res Paed 2015;83:376-89.
15. Coviello AD, Legro RS, Dunaif A. Adolescent girls with polycystic ovary syndrome have an increased risk of the metabolic syndrome associated with increasing androgen levels independent of obesity and insulin resistance. J Clin Endocrinol Metab 2006;91:492-7.
16. Vrbíková J, Zamrazilová H, Sedláčková B, Šnajderová M. Metabolic syndrome in adolescents with polycystic ovary syndrome. Gynecol Endocrinol 2011;27:820-2.
17. Rossi B, Sukalich S, Droz J, Griffin A, Cook S, Blumkin A, *et al*. Prevalence of metabolic syndrome and related characteristics in obese adolescents with and without polycystic ovary syndrome. J Clin Endocrinol Metab 2008;93:4780-6.
18. Barber TM, Golding SJ, Alvey C, Wass JA, Karpe F. Global adiposity rather than abnormal regional fat distribution characterizes women with polycystic ovary syndrome. J Clin Endocrinol Metab 2008;93:999-1004.
19. Khadilkar VV, Khadilkar AV. Revised Indian academy of pediatrics 2015 growth charts for height, weight and body mass index for 5-18-year-old Indian children. Indian J Endocrinol Metab 2015;19:470-6.
20. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril 2004;81:19-25.
21. Ferriman D, Gallwey JD. Clinical assessment of body hair growth in women. J Clin Endocrinol Metab 1961;21:1440-7.
22. Khadilkar A, Ekbote V, Chiplonkar S, VamanKhadilkar V, Kajale N, Kulkarni S, *et al*. Waist circumference percentiles in 2-18 year old Indian children. J Pediatr 2014;164:1358-62.e2.
23. Shah A, Sarin M, Karunanand B, Mohapatra SC, Bhat S A. Association of hormonal status with anthropometric and biochemical parameters in

women with polycystic ovarian syndrome. J Community Health Manag 2017;4:30-4.
24. Azziz R, Kashar-Miller MD. Family history as a risk factor for the polycystic ovary syndrome. J Pediatr Endocrinol Metab 2000;13:1303-6.
25. Kahsar-Miller MD, Nixon C, Boots LR, Go RC, Azziz R. Prevalence of polycystic ovary syndrome (PCOS) in first-degree relatives of patients with PCOS. Fertil Steril 2001;75:53-8.
26. Goran M, Gower BA. Longitudinal study on pubertal insulin resistance. Diabetes 2001;50:2444-50.
27. Cree-Green M. Worldwide dissatisfaction with the diagnostic process and initial treatment of PCOS. J Clin Endocrinol Metab 2017;102:375-8.
28. Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, *et al*. Androgen excess society. Position statement: Criteria for defining pcos as a predominantly hyperandrogenic syndrome: An Androgen excess society guideline. J Clin Endocrinol Metab 2006;91:4237-45.

Funding: None; Conflicts of Interest: None Stated.

How to cite this article: Muthukumar A, Krishna VG, Prasad HK, Narayanaswamy K, Ravisekar CV, Krishnamoorthy N. Clinical, biochemical and radiological profile of polycystic ovary syndrome in adolescents attending an obesity clinic. Indian J Child Health. 2021; 8(1):20-25.