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ORIGINAL ARTICLE

Correlation of Clinical, Hormonal, Biochemical and Ultrasound Parameters Between Adult and Adolescent Polycystic Ovarian Syndrome

Adult and Adolescent PCOS

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Abstract

Purpose To correlate the clinical, hormonal, biochemical and ultrasound parameters in adolescent patients with polycystic ovarian syndrome (PCOS) and to compare them with adult patients.

Methods This was a prospective, correlational study. 50 adult (20–35 years) and 50 adolescent patients (15–19 years) who had features of PCOS (Rotterdam Criteria, 2003) were selected. The control group comprised of 50 women of same age in each group with normal parameters. Pelvic ultrasound was done in early follicular phase (3–5th day of menstrual cycle). Assessment of hormonal and biochemical parameters (LH/FSH ratio, free testosterone level, lipid profile and fasting glucose/insulin ratio) and grey-scale ultrasound was done.

Results No significant difference was observed in menstrual pattern in adults and adolescents with PCOS. The mean values of serum LH/FSH ratio and free testosterone were significantly higher in both adult and adolescent PCOS patients as compared to their controls ($p < 0.001$). The mean value of serum insulin was significantly higher ($p < 0.001$) with positive correlation (adult: $r = 0.655$, $p < 0.01$; adolescent: $r = 0.451$, $p < 0.01$) of serum insulin with free testosterone. Hyperandrogenemia without hyperinsulinemia was found in 56% adolescent and 60% adult PCOS patients. 82% adolescent and 88% adult PCOS patients showed multiple follicles (> 5) on ultrasound. The ovarian morphology had positive correlation with serum LH and free testosterone. The mean ovarian volume was significantly higher in adult (10.48 ± 4.38 vs. 4.17 ± 0.91) and adolescent (11.08 ± 5.82 vs. 4.23 ± 0.89) PCOS patients, when compared with controls, respectively.

Conclusion No statistically significant difference was noted in PCOS between adults and adolescents.

Keywords Adult PCOS · Adolescent PCOS · Pelvic ultrasound · Hyperinsulinemia · Hyperandrogenism

Introduction

Polycystic ovarian syndrome (PCOS) is one of the most common human endocrinopathies, affecting 6–10% of women of reproductive age [1]. PCOS is characterized by hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology on ultrasound [2]. Out of the three features, hyperandrogenism is the most consistent feature of PCOS both in adults and adolescents [3]. The signs and symptoms of PCOS during adolescence are often overlapped by characteristics of normal puberty. It is also associated with long-term significant morbidity in terms of metabolic syndrome, impaired reproductive function, psychosocial problems and increased risk of cancer [4].

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The majority of adolescent patients with menstrual cycle disorder and high LH concentration continue to suffer from ovulatory disorder during adulthood [5]. Hereby, we have compared the clinical, hormonal, biochemical and ultrasound parameters of adolescent PCOS patients with that of adult PCOS.

Material and Methods

The study was done after approval of institutional ethical committee and was in accordance with the 1964 Declaration of Helsinki and its later amendments. Two groups of patients with PCOS, 50 adults and 50 adolescents were part of this prospective, correlational study. Two groups of healthy volunteers, one for adult (age group 20–35 years, $n=50$) and another for adolescents (age group 15–19 years, $n=50$, menarche 2 years or more before the study) were taken as controls. These groups were further divided on the basis of body mass index (BMI) into obese (BMI > 27 kg/m^2) and non-obese (BMI ≤ 27 kg/m^2).

Inclusion Criteria

Patients having oligomenorrhoea (> 35 days cycle length) or amenorrhoea (absent menstruation for last three months), hirsutism, acne or seborrhoea (modified Ferriman and Gallwey Score ≥ 8), [6] LH/FSH ratio > 2, ultrasound (transabdominal/transvaginal) features suggestive of PCOS were included. The diagnosis of PCOS was based on Rotterdam criteria with the presence of at least two out of three findings (i) oligo- or anovulation (ii) clinical and/or biochemical signs of hyperandrogenism (iii) polycystic ovaries on ultrasound— the presence of ≥ 12 follicles, 2–9 mm in diameter, and/or increased ovarian volume (> 10 ml) [7].

Exclusion Criteria

Patients with Cushing's syndrome, congenital adrenal hyperplasia, hypothyroidism or hyperthyroidism, adrenal or ovarian tumours or hyperprolactinaemia and women on oral contraceptives for last four months were excluded.

The women in the control group had regular menstruation, normal LH/FSH ratio, no hirsutism and normal pelvic ultrasound. All tests were performed in early follicular phase (cycle days 3–5) in controls and oligomenorrhoeic patients while at random in cases with amenorrhea. BMI was calculated as weight (kg) divided by height (m^2). Waist–hip ratio (WHR) was calculated from the circumference measured in duplicate in supine position (waist: midway between the lower rib margin and the iliac crest, hip: widest circumference over the greater trochanters). Blood samples were taken after overnight fasting on the day of ultrasound. Serum LH, FSH, T_3, T_4, TSH, prolactin and free testosterone estimation was done by immunoradiometric assay by using coat-A count/RMA kit DPC Mumbai. Serum insulin estimation was done by using BARC, RIA Kit Mumbai. Blood glucose estimation was done by autoanalyzer. Pelvic ultrasound was performed using 2–5 MHz transabdominal and 5–9 MHz transvaginal transducers (Diagnostic Ultrasound, iU22—Philips Medical system, California, the USA). Ovarian volume was calculated using simplified formula for prolate ellipsoid (0.5 × length × width × thickness) [8].

Ovarian roundness index was expressed as the ratio between the second-largest diameter (in width) over the maximum diameter (length). Ovarian stromal echogenicity was scored as normal (score 1), moderately increased (score 2) and markedly increased (score 3). The number of follicles was established with scanning of each ovary from the inner margin to the outer margin in the longitudinal cross section depending upon the longitudinal diameter of the follicle. The size of the follicle was calculated from the mean of two diameters (longitudinal and anteroposterior).

Statistical Analysis

The values were expressed as mean ± standard deviation. The correlation was analyzed using Karl's Pearson correlation coefficient (r). $P<0.05$ was considered significant. All the analysis was done using SPSS-20 version for Windows.

Results

The mean BMI of adult PCOS patients and their age-matched controls was 26.14 ± 3.38 kg/m^2 and 26.13 ± 3.61 kg/m^2, respectively. Similarly, the mean BMI of adolescent PCOS patients and their controls was 27.10 ± 4.55 kg/m^2 and 26.22 ± 2.44 kg/m^2, respectively (Table 1).

In adolescent PCOS patients, oligomenorrhoea was the most common (62%) presenting complaint followed by Android obesity (60%), obesity (58%) and hirsutism (42%). In adult PCOS patients, oligomenorrhoea (82%), primary infertility (76%), Android obesity (48%), obesity (46%) and acne (40%) were the main complaints.

Different endocrinological parameters in adult and adolescent PCOS patients and their controls showed the mean value of serum LH, LH/FSH ratio and free testosterone to be significantly higher ($p<0.01$), both in adults and adolescents as compared to their controls. However, when these values were compared between the adult and adolescent patients, there was statistically insignificant difference (Table 1).

Hyperinsulinemia was present in PCOS patients ($p<0.001$ in adults and <0.01 in adolescents) with, however,

Table 1 Comparison of physical, hormonal and biochemical parameters in adult and adolescent PCOS patients and their controls

Variables	Adults		Adolescents		Significance of difference (mean difference)		
	Control $N=50$	Case $N=50$	Control $N=50$	Case $N=50$	Adult case Vs. control	Adolescent case Vs. control	Adult case Vs. Adolescent case
Age (yr.)	24.04 ± 1.88	24.10 ± 2.04	16.38 + 1.27	16.18 + 1.48	NS	NS	NS
BMI (Kg/m^2)	26.13 ± 3.61	26.14 ± 3.38	26.22 + 2.44	27.10 + 4.55	NS	NS	NS
Waist–hip ratio	0.83 ± 0.03	0.85 ± 0.05	0.83 + 0.06	0.86 + 0.04	0.02*	0.03*	0.01^{NS}
LH (m IU/ml)	5.00 ± 1.36	8.08 ± 2.84	5.06 + 1.55	7.75 + 4.6	3.08**	2.69*	0.32^{NS}
FSH (m IU/ml)	4.48 ± 1.05	4.07 ± 1.13	4.06 + 1.17	3.88 + 1.21	0.41^{NS}	0.18^{NS}	0.19^{NS}
LH/FSH ratio	1.14 ± 0.27	2.24 ± 0.44	1.28 + 0.26	2.08 + 1.27	1.10**	0.80*	0.16^{NS}
TSH (μU/ml)	3.08 ± 0.88	3.31 ± 0.52	2.92 + 0.983	3.08 + 0.89	0.23^{NS}	0.16^{NS}	0.24^{NS}
PRL (ng/ml)	13.40 ± 1.44	13.51 ± 1.58	14.02 + 1.50	13.76 + 1.29	0.11^{NS}	0.23^{NS}	0.28^{NS}
Free testosterone (pg/ml)	1.67 ± 0.15	3.52 ± 0.54	1.50 + 0.34	3.60 + 0.36	1.85**	2.10**	0.07^{NS}
Fasting glucose (mg/dL)	91.11 ± 2.69	92.70 ± 6.13	89.75 + 2.64	91.55 + 6.45	1.59^{NS}	1.82^{NS}	1.19^{NS}
Fasting insulin (μU/ml)	11.88 ± 1.43	24.32 ± 4.37	11.69 + 1.47	25.83 + 4.74	12.44***	14.19***	1.51^{NS}
Glucose/insulin ratio	7.77 ± 0.938	3.92 ± 0.739	7.79 + 1.03	3.66 + 0.755	3.85***	4.13***	0.26^{NS}

*$p<0.05$, **$p<0.01$, ***$p<0.001$

no statistical difference in fasting blood sugar level. The glucose/insulin ratio was statistically low along with increased fasting insulin in both groups of PCOS patients as compared to their controls showing evidence of insulin resistance. No statistically significant difference was observed between adolescents and adults.

Similarly, when serum lipid profiles of adult and adolescent PCOS patients were compared with BMI-matched controls, there was no statistical difference ($p>0.05$) (Table 2). There was a significant positive ($p<0.01$) correlation of serum LDL (adult: $r=0.809$, $p<0.01$; adolescent: $r=0.323$, $p<0.05$), triglycerides (adult: $r=0.610$, $p<0.01$; adolescent: $r=0.645$, $p<0.01$) and cholesterol (adult: $r=0.456$, $p<0.01$; adolescent: $r=0.373$, $p<0.01$) with BMI, while serum HDL negatively correlated (adult: $r=-0.577$, $p<0.01$; adolescent: $r=-0.530$, $p<0.01$) with BMI in both adult and adolescent PCOS patients (Table 3). At the same time, there was no correlation of serum-free testosterone with lipid profile in both adults and adolescents. Serum-free testosterone positively correlated (statistically significant) with WHR (>0.85) in both adult ($r=0.396$, $p<0.01$) and adolescent ($r=0.471$, $p<0.01$) PCOS patients (Table 4).

Comparative study of ultrasonographic parameters in adult and adolescent PCOS patients showed significantly higher ($p<0.001$) values of mean ovarian volume and mean number of follicles with increased stromal echogenicity in both adult and adolescent PCOS patients on comparison with their controls. The roundness index was statistically not

Table 2 Comparison of serum lipid profiles in adult and adolescent obese PCOS patients and controls

Serum Lipids	Adult Obese ($N=48$)			Adolescent Obese ($N=48$)		
	Control ($N=25$)	Case ($N=23$)	Significance of difference (Mean Difference)	Control ($N=25$)	Case ($N=23$)	Significance of difference (Mean Difference)
Cholesterol (mg/dl)	152.13 ± 14.78	147.03 ± 13.23	5.10 ^{NS}	158.27 ± 11.12	153.40 ± 12.58	4.87^{NS}
VLDL (mg/dl)	26.47 ± 2.57	25.27 ± 2.03	1.20 ^{NS}	26.60 ± 2.23	25.88 ± 2.80	0.72 ^{NS}
LDL (mg/dl)	88 ± 8.90	86.43 ± 12.86	1.57 ^{NS}	96.54 ± 6.41	99.19 ± 4.48	2.65 ^{NS}
Triglycerides (mg/dl)	131.24 ± 11.65	124.85 ± 10.48	6.39 ^{NS}	156.36 ± 12.88	160.35 ± 16.36	3.99 ^{NS}
HDL (mg/dl)	42.76 ± 3.25	40.86 ± 3.94	1.90 ^{NS}	41.48 ± 2.32	42.87 ± 3.97	1.39 ^{NS}

NS—$p>0.05$

Table 3 Correlation of serum testosterone levels and body mass index with lipid profile in adult and adolescent PCOS patients

Serum lipids	Adult PCOS		Adolescent PCOS	
	Free testosterone	Body mass index	Free testosterone	Body mass index
Cholesterol (mg/dl)	0.194^{NS}	0.373**	0.056^{NS}	0.456**
Triglycerides (mg/dl)	0.209 ^{NS}	0.645	0.137 ^{NS}	0.610**
VLDL (mg/dl)	0.214 ^{NS}	0.203**	0.148 ^{NS}	0.092
LDL (mg/dl)	0.206 ^{NS}	0.323	0.147 ^{NS}	0.809**
HDL (mg/dl)	− 0.142 ^{NS}	− 0.530**	− 0.241 ^{NS}	− 0.577**

*$p < 0.05$; **$p < 0.01$

Table 4 Correlation coefficient (r) of ovarian morphology with different hormonal parameters in adult and adolescent PCOS patients

Correlation	Adult PCOS (r)	Adolescent PCOS(r)
Ovarian volume & LH	0.495**	0.525**
Ovarian volume & Free testosterone	0.401**	0.569**
Mean follicular number & free testosterone	0.429**	0.627**
Mean follicular number & LH	0.343*	0.304*
LH & free testosterone	0.403**	0.498**
Fasting insulin & free testosterone	0.655**	0.451**
Waist–hip ratio vs. free testosterone	0.396**	0.471**
Waist–hip ratio vs. insulin	0.584**	0.562**

*$p < 0.05$; **$p < 0.01$

significant when compared with the controls. However, no significant difference was seen when adults were compared with adolescents (Table 5).

Discussion

Polycystic ovarian syndrome is a heterogenous clinical entity characterized by signs and symptoms of hyperandrogenism and anovulatory disorder often associated with infertility and obesity [9] The accepted aetiologies include disordered neuroendocrine gonadotrophin secretion, hyperandrogenism, insulin resistance and hyperinsulinism or their combination [10]. PCOS affects both adults as well as adolescents. High serum LH with increased androgens in oligomenorrheic adolescents is functional step of maturation of the ovulatory system. The high concentration of these hormones in oligomenorrheic girls is not transient and may be an early sign of PCOS [11]. These adolescents with oligomenorrhoea continue to have this menstrual cycle pattern combined with subfertility in adulthood. Thus, complete evaluation of adolescent girls should be considered before reassuring them or prescribing oral contraceptives.

Table 5 Comparison of ultrasonographic parameters in adult and adolescent patients and their controls

Ultrasound parameters	Adults		Adolescents		Significance of difference		
	Control $N=50$	Case $N=50$	Control $N=50$	Case $N=50$	Adult case vs control	Adolescent case vs control	Adult case vs Adolescent case
Mean ovarian volume (ml)	4.17 ± 0.91	10.48 ± 4.38	4.23 ± 0.89	11.08 ± 5.82	6.84***	6.31***	0.60^{NS}
Mean number of follicles							NS
0–4	100%	18%	100%	12%	$p < 0.0$	$p < 0.05$	
5–11	0	8%	0	16%	$P < 0.05$	$P < 0.05$	
> 12	0	74%	0	72%	$P < 0.001$	$P < 0.001$	
Stromal echogenicity (score)%							
1	100%	0	100%	0	$p < 0.0$	$P < 0.05$	NS
2	0	18%	0	12%	$P < 0.001$	$P < 0.001$	
.3	0	82%	0	88%	$P < 0.05$	$P < 0.05$	
Roundness index	0.58 ± 0.03	0.57 ± 0.04	0.52 ± 0.05	0.53 ± 0.07	0.011^{NS}	0.003^{NS}	0.01^{NS}

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$

The clinical profile showed that most of these PCOS patients were suffering from oligomenorrhoea (adolescent 62%, adult 80%), infertility (76%), hirsutism (adolescent 42%, adult 40%) and acne (adolescent 26%, adult 24%). All these symptoms were due to chronic anovulation and hyperandrogenism, the basic pathophysiology in PCOS [12]. Azziz et al. reported the incidence of hirsutism in 60% of their patients and also found the menstrual irregularity to be a very common manifestation of adult PCOS (> 75%) [13]. Although mild hirsutism is very common in early adolescent age just after menarche, a moderate to severe hirsutism or persistent acne resistant to topical oestrogen therapy was labelled as a diagnostic criterion of hyperandrogenism by Kamboj et al. [14]. They also recommended the three parameters; 1) consecutive menstrual interval > 90 days even in the first year after menarche, 2) menstrual interval persisting of < 21 or > 45 days two or more years after menarche and 3) lack of menses by 15 years of age or after 2–3 years of breast budding as evidence of true ovulatory dysfunction.

Franks reported that 50% of the PCOS patients were obese (BMI ≥ 28 kg/m^2)[15]. We observed that 29 out of 50 adolescent (58%) and 23 out of 50 adult (46%) PCOS patients were obese (BMI > 27 kg/m^2). Acanthosis nigricans, obesity including truncal obesity, dyslipidemia, hypertension, glucose intolerance have been found in 5% of adolescents' general population [16]. Obesity is said to be a better predictor of metabolic dysfunction than PCOS itself [17] Androgen excess may increase the risk of developing metabolic syndrome independent of obesity [16]. In our study, 76% of adolescent and 64% of adult PCOS patients showed biochemical evidence of LH/FSH ≥ 2. Increased GnRH pulse frequency followed by rise in LH β subunit without affecting FSH β subunit was responsible for higher LH/FSH ratio in PCOS patients [17]. The increased LH pulse frequency and amplitude along with the acceleration of the GnRH pulse generator have been suggested to be an intrinsic defect of PCOS in peripubertal girls with ovarian hyperandrogenism [18].

The measurement of total and/or free testosterone has been the most recommended hormonal test to diagnose hyperandrogenemia [19]. The increased testosterone levels in our PCOS patients might be due to hyperinsulinemia resulting in decreased sex hormone-binding globulin under the effect of central obesity (WHR > 0.85). The effect of central obesity on androgen production was also supported in our study as serum-free testosterone positively correlated significantly with WHR (> 0.85) in both adult ($r = 0.396$, $p < 0.01$) and adolescent ($r = 0.471$, $p < 0.01$) PCOS patients. Hyperandrogenism subgroup with PCOS in adolescents showed increased incidence of insulin resistance and inflammation [16]. Free testosterone positively correlated with hyperinsulinemia in both adult ($r = 0.655$, $p < 0.01$) and adolescent ($r = 0.451$, $p < 0.01$) PCOS patients, it was believed that this hyperinsulinemia was responsible for hyperandrogenism [20]. The mechanism of hyperinsulinemia-induced hyperandrogenism might be due to inhibition of hepatic synthesis of SHBG or due to increased androgen production by theca cells by activating IGF – 1 receptors [21]. Other researchers also found positive correlation between hyperinsulinemia and hyperandrogenism as in our study [21]. The fasting serum insulin was significantly higher in our PCOS patients, both adults and adolescents ($p < 0.01$), without any significant difference in fasting blood sugar level ($p > 0.05$). Fasting insulin level of > 20μU/ mL alone is an indicator of insulin resistance, although in some populations, fasting glucose/insulin ratio < 4.5 is said to be more than 90% sensitive indicator of insulin resistance [22]. The glucose/insulin ratio (< 4.5) was significantly lower in our PCOS patients in both adults and adolescents. However, the homeostatic model assessment (HOMA), a product of fasting glucose and insulin level divided by a constant, is also a reflector of insulin resistance, but it may miss 50% of the young PCOS women with insulin resistance [23]. Both insulin resistance and hyperandrogenemia have been found to be more severe in adolescents with PCOS as compared to general adolescent population [24]. It is said that there is 50% decrease in insulin sensitivity in normal healthy adolescents with compensatory hyperinsulinemia which reverts back to normal pre-pubertal level in adulthood in the absence of PCOS [25].

However, there was no significant difference in serum lipid profiles when the obese PCOS patients were compared to their BMI-matched controls ($p > 0.05$, Table 3). Thus, dyslipidemia found in our PCOS patients might be due to obesity and not due to the disease itself. The excess adiposity itself is supposed to suppress ovulation via LH suppression and causes hyperandrogenemia (via adipose tissue formation of testosterone from androstenedione) [26]. It might also be due to insulin resistance that promotes release of non-esterified fatty acids from liver and adipose tissue due to decreased lipoprotein-lipase activity that contributes to dyslipidemia. Our results were also consistent with those obtained by others [16]. However, we found no correlation between serum-free testosterone and lipid profile. This might be explained by the fact that PCOS is a hyperestrogenic state and higher oestrogen might balance the effect of androgens on serum lipids.

The mean ovarian volume was significantly increased in PCOS patients when compared to controls. In our study, only 37 out of 50 (74%) adult PCOS patients and 36 out of 50 (72%) adolescent PCOS patients had significantly increased ovarian volume. This increased ovarian volume in our PCOS patients could be due to theca cell hyperplasia and increased rate of follicular atresia causing polycystic transformation of ovaries under the effect of androgen [27]. Others have found the ovarian volume to correlate with serum insulin but not with the circulating androgens, LH,

FSH and BMI [28]. Here, we should not forget the fact that ultrasound has technical problems in adolescent population and in obese patients. The ovarian appearance, as well as the volume, both may vary during adolescence such that the ovaries may not only show polycystic morphology over time, but also the enlarged ovaries with polycystic appearance can subsequently become normal [29]. Moreover, 10–48% of adolescent population who do not have PCOS may have polycystic appearance of ovaries [30].

In our study, mean ovarian volume positively correlated with serum LH and free testosterone in PCOS patients. This correlation explained that enlarged ovarian volume was under the effect of high LH and testosterone levels. Some also found similar correlation of ovarian volume with LH ($r=0.30$, $p=0.003$) and testosterone ($r=0.34$, $p=0.001$) [31]. However, our results showed 7 out of 50 adolescent PCOS (14%) patients and 10 out of 50 adult PCOS (20%) patients had increased ovarian volume without LH/FSH ratio ≥ 2. In these patients, increased ovarian volume might be due to LH independent effect of testosterone on ovarian volume.

The polycystic transformation of ovaries might be due to continuous stimulation of follicles but not to the point of full maturation and ovulation because the FSH levels were not totally depressed. Balen strongly recommended more than 10 follicles as the diagnostic criteria for PCOS [32]. The mean number of follicles more significantly correlated with free testosterone rather than LH in our study. This might be due to independent effect of androgen on increased rate of atresia with consecutive augmentation of ovarian volume leading to polycystic transformation of ovaries.

Similar to Battaglia et al. [33], stromal echogenicity was significantly increased in both the groups which could be due to hyperplasia of theca cells and increased formation subsequent to the excessive follicular maturation and atresia.

Conclusions

PCOS is a complex multifaceted disorder and should be diagnosed using the same criteria in adults as well as in adolescents and we should start early with meaningful intervention to avoid long-term morbid complications, especially in adolescents.

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Declarations

Conflict of interest The authors declare that they have no conflict of interest.

Human and Animal Rights This article does not contain any studies with animal subjects.

Ethical Approval The study was performed after approval of the institutional ethical committee of the Institute of Medical Sciences, Banaras Hindu University, Varanasi, India. All procedures performed in the study involving human participants were in accordance with the ethical standards of the institutional ethical committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. This article does not contain any studies with animals performed by any of the authors.

Informed Consent Informed consent was obtained from all the individual participants included in the study.

References

1. Bozdag G, Mumusoglu S, Zengin D, et al. The prevalence and phenotypic features of polycystic ovary syndrome: a systematic review and meta-analysis. Hum Reprod. 2016;31(12):2841–55. https://doi.org/10.1093/humrep/dew218.
2. Teede HJ, Misso ML, Costello MF, et al. Recommendations from the international evidence-based guideline for the assessment and management of polycystic ovary syndrome. Clin Endocrinol (Oxf). 2018;89(3):251–68. https://doi.org/10.1111/cen.13795.
3. Ibáñez L, Oberfield SE, Witchel S, et al. An International Consortium Update: Pathophysiology, Diagnosis, and Treatment of Polycystic Ovarian Syndrome in Adolescence. Horm Res Paediatr. 2017;88(6):371–95. https://doi.org/10.1159/000479371.
4. Hart R, Doherty DA. The potential implications of a PCOS diagnosis on a woman's long-term health using data linkage. J Clin Endocrinol Metab. 2015; 100(3): 911–9. doi: https://doi.org/10.1210/jc.2014-3886. Epub 2014 Dec 22. Erratum in: J Clin Endocrinol Metab. 2015; 100(6): 2502. PMID: 25532045.
5. Kimura K, Minakami H, Tamada T. A longitudinal study on the prognosis of ovulatory disturbance in teenage patients with high LH and normal FSH serum levels. Nihon Naibunpi Gakkai Zasshi. 1988;64(10):1088–101.
6. Sirmans SM, Pate KA. Epidemiology, diagnosis, and management of polycystic ovary syndrome. Clin Epidemiol. 2013;6:1–13. https://doi.org/10.2147/CLEP.S37559.PMID:24379699;PMCID:PMC3872139.
7. 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(1):19–25. https://doi.org/10.1016/j.fertnstert.2003.10.004.
8. Swanson M, Sauerbrei EE, Cooperberg PL. Medical implications of ultrasonically detected polycystic ovaries. J Clin Ultrasound. 1981;9(5):219–22. https://doi.org/10.1002/jcu.1870090504.
9. Rosenfield RL, Ehrmann DA. The Pathogenesis of Polycystic Ovary Syndrome (PCOS): The Hypothesis of PCOS as Functional Ovarian Hyperandrogenism Revisited. Endocr Rev. 2016;37(5):467–520.
10. Dumesic DA, Oberfield SE, Stener-Victorin E, et al. Scientific Statement on the Diagnostic Criteria, Epidemiology, Pathophysiology, and Molecular Genetics of Polycystic Ovary Syndrome. Endocr Rev. 2015;36(5):487–525. https://doi.org/10.1210/er.2015-1018.PMID:26426951;PMCID:PMC4591526.
11. Rosenfield RL, Ehrmann DA, Littlejohn EE. Adolescent polycystic ovary syndrome due to functional ovarian hyperandrogenism persists into adulthood. J Clin Endocrinol Metab. 2015;100(4):1537–43.

12. Rosenfield RL. The Polycystic Ovary Morphology-Polycystic Ovary Syndrome Spectrum. J Pediatr Adolesc Gynecol. 2015;28(6):412–9.
13. Azziz R, Carmina E, Dewailly D, et al. Positions statement: criteria for defining polycystic ovary syndrome as a predominantly hyperandrogenic syndrome: an Androgen Excess Society guideline. J Clin Endocrinol Metab. 2006;91(11):4237–45. https://doi.org/10.1210/jc.2006-0178.
14. Kamboj MK, Bonny AE. Polycystic ovary syndrome in adolescence: diagnostic and therapeutic strategies. Transl Pediatr. 2017;6(4):248–55. https://doi.org/10.21037/tp.2017.09.11.PMID:29184806;PMCID:PMC5682369.
15. Franks S. Polycystic ovary syndrome: a changing perspective. Clin Endocrinol (Oxf). 1989;31(1):87–120. https://doi.org/10.1111/j.1365-2265.1989.tb00457.x.
16. Alemzadeh R, Kichler J, Calhoun M. Spectrum of metabolic dysfunction in relationship with hyperandrogenemia in obese adolescent girls with polycystic ovary syndrome. Eur J Endocrinol. 2010;162(6):1093–9. https://doi.org/10.1530/EJE-10-0205.
17. Fulghesu A, Magnini R, Portoghese E, et al. Obesity-related lipid profile and altered insulin incretion in adolescents with polycystic ovary syndrome. J Adolesc Health. 2010;46(5):474–81. https://doi.org/10.1016/j.jadohealth.2009.10.008.
18. Apter D, Bützow T, Laughlin GA, et al. Accelerated 24-hour luteinizing hormone pulsatile activity in adolescent girls with ovarian hyperandrogenism: relevance to the developmental phase of polycystic ovarian syndrome. J Clin Endocrinol Metab. 1994;79(1):119–25. https://doi.org/10.1210/jcem.79.1.8027216.
19. Escobar-Morreale HF, Carmina E, Dewailly D, et al. Epidemiology, diagnosis and management of hirsutism: a consensus statement by the Androgen Excess and Polycystic Ovary Syndrome Society. Hum Reprod Update. 2012;18(2):146–70.
20. Elkind-Hirsch KE, Valdes CT, McConnell TG, et al. Androgen responses to acutely increased endogenous insulin levels in hyperandrogenic and normal cycling women. Fertil Steril. 1991;55(3):486–91.
21. Buyalos RP, Geffner ME, Bersch N, et al. Insulin and insulin-like growth factor-I responsiveness in polycystic ovarian syndrome. Fertil Steril. 1992;57(4):796–803.
22. Legro RS, Castracane VD, Kauffman RP. Detecting insulin resistance in polycystic ovary syndrome: purposes and pitfalls. Obstet Gynecol Surv. 2004;59(2):141–54. https://doi.org/10.1097/01.OGX.0000109523.25076.E2.
23. Fulghesu AM, Angioni S, Portoghese E, et al. Failure of the homeostatic model assessment calculation score for detecting metabolic deterioration in young patients with polycystic ovary syndrome. Fertil Steril. 2006;86(2):398–404. https://doi.org/10.1016/j.fertnstert.2006.01.024.
24. Lewy VD, Danadian K, Witchel SF, et al. Early metabolic abnormalities in adolescent girls with polycystic ovarian syndrome. J Pediatr. 2001;138(1):38–44. https://doi.org/10.1067/mpd.2001.109603.
25. Hannon TS, Janosky J, Arslanian SA. Longitudinal study of physiologic insulin resistance and metabolic changes of puberty. Pediatr Res. 2006;60(6):759–63. https://doi.org/10.1203/01.pdr.0000246097.73031.27.
26. Rosenfield RL, Bordini B. Evidence that obesity and androgens have independent and opposing effects on gonadotropin production from puberty to maturity. Brain Res. 2010;1364:186–97.
27. Diamanti-Kandarakis E, Dunaif A. Insulin resistance and the polycystic ovary syndrome revisited: an update on mechanisms and implications. Endocr Rev. 2012;33(6):981–1030.
28. Reid SP, Kao CN, Pasch L, et al. Ovarian morphology is associated with insulin resistance in women with polycystic ovary syndrome: a cross sectional study. Fertil Res Pract. 2017;3:8. https://doi.org/10.1186/s40738-017-0035-z.PMID:28620546;PMCID:PMC5450099.
29. Venturoli S, Porcu E, Fabbri R, et al. Longitudinal change of sonographic ovarian aspects and endocrine parameters in irregular cycles of adolescence. Pediatr Res. 1995;38(6):974–80. https://doi.org/10.1203/00006450-199512000-00024.
30. Blank SK, Helm KD, McCartney CR, et al. Polycystic ovary syndrome in adolescence. Ann N Y Acad Sci. 2008;1135:76–84. https://doi.org/10.1196/annals.1429.005.
31. Pache TD, Chadha S, Gooren LJ, et al. Ovarian morphology in long-term androgen-treated female to male transsexuals. A human model for the study of polycystic ovarian syndrome? Histopathology. 1991;19(5):445–52.
32. Balen AH, Conway GS, Kaltsas G, et al. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod. 1995;10(8):2107–11. https://doi.org/10.1093/oxfordjournals.humrep.a136243.
33. Battaglia C, Artini PG, D'Ambrogio G, et al. The role of color Doppler imaging in the diagnosis of polycystic ovary syndrome. Am J Obstet Gynecol. 1995;172(1 Pt 1):108–13. https://doi.org/10.1016/0002-9378(95)90094-2.

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