International Journal of Reproduction, Contraception, Obstetrics and Gynecology
Singh N et al. Int J Reprod Contracept Obstet Gynecol. 2022 Aug;11(8):2180-2183
www.ijrcog.org
pISSN 2320-1770 | eISSN 2320-1789

DOI: https://dx.doi.org/10.18203/2320-1770.ijrcog20221933

Original Research Article

Comparison of the various diagnostic criteria used in polycystic ovary syndrome

Neha Singh, Nupur Hooja*, Angela Yadav, Pooja Bairwa, Aditi Jaiswal

Department of Obstetrics and Gynecology, SMS Medical College, Jaipur, Rajasthan, India

Received: 29 May 2022
Revised: 29 June 2022
Accepted: 30 June 2022

***Correspondence:**
Dr. Nupur Hooja,
E-mail: nupurhooja@gmail.com

ABSTRACT

Background: Polycystic Ovary Syndrome (PCOS) represents one of the major causes of infertility in women. Various criteria are set to diagnose PCOS, some over diagnose and some underdiagnose it. The objective of the study was to compare the various criteria used for the diagnosis of PCOS: NIH 1990 criteria, Rotterdam 2003 criteria and AE-PCOS Society 2006 criteria.
Methods: This was a cross-sectional study conducted over a period of one year. Patients with suspicion of PCOS were selected. After complete history and examination, blood samples were collected and androgen levels were measured. They were labeled as PCOS based on their fulfillment of the criteria as per Rotterdam, NIH and AE-PCOS Society.Data was analysed and conclusions drawn.
Results: Of the participants, 25.7%, 28.5%, and 62.8% were diagnosed with PCOS using NIH, AE-PCOS Society, and Rotterdam criteria, respectively. Phenotypes that included hyperandrogenism and/or hyperandrogenemia as part of their criteria showed increase in values of DHEAS and S. testoterone as compared to the normoandrogenic phenotype included in only the Rotterdam criteria.
Conclusions: It is crucial to establish the diagnostic criteria for PCOS and initiate early treatment as this may play a role in the prevention of metabolic and cardiovascular diseases.

Keywords: Hyperandrogenism, Polycystic ovary syndrome, Rotterdam

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a heterogeneous and complex disorder that has both metabolic and hormonal implications and represents one of the major causes of infertility in women.[1] Although PCOS is heralded as one of the most common endocrine disorders occurring in women, its diagnosis, management, and associated long-term health risks remain controversial. The various criteria used for the diagnosis of PCOS are: National Institute of Health 1990 criteria, Rotterdam 2003 criteria and Androgen Excess-PCOS Society 2006 criteria. The objective of the study was to compare the various criteria used for the diagnosis of PCOS.

METHODS

This was a hospital based descriptive study conducted in the Department of Obstetrics and Gynaecology, SMS Medical College, Jaipur from July 2020 till June 2021.

Seventy women, aged 18-40 year with symptoms suggestive of Polycystic Ovary Syndrome: oligo/anovulation, hyperandrogenism clinical (hirsutism or less commonly, male pattern alopecia) or biochemical (raised Free Androgen Index or free testosterone) and polycystic ovaries on ultrasound were included in the study.

Women who gave history of inflammatory diseases like rheumatoid arthritis, chronic obstructive lung disease, chronic liver disease or those with non-classical congenital adrenal hyperplasia (NC-CAH), Cushing's syndrome, androgen-secreting tumors, hyperprolactinemia, thyroid diseases, drug-induced androgen excess, as well as other causes of oligomenorrhea or anovulation were not included. Pregnant or lactating women were also excluded.

Diagnosis of PCOS as per the NIH criteria (1990) required hyperandrogenism and or hyperandrogenemia and anovulation or oligo-ovulation. Hence, based on this, three principal phenotypes were identified (1) hirsutism, hyperandrogenemia and oligo-ovulation (2) hirsutism and oligo-ovulation (3) hyperandrogenemia and oligo-ovulation.[2] Hyperandrogenemia was defined by testosterone (T) and/or free testosterone (FT) and/or dihydroandrostenedione (DHEA) higher than 75% of the upper limits of each hormone.[3] Hirsutism, defined by the FG score (Ferriman - Gallwey) is the presence of excessive facial hairs on the side of the face, upper lip, chin and it is also observed in chest region in severe PCOS.[4]

As per the Rotterdam criteria (2003) two of the three must be present: Anovulation or oligo-ovulation, clinical or biochemical signs of hyperandrogenism, PCOM (PCO Morphology) Hence, it added two new phenotypes of PCOS: patients who have PCOM, hirsutism and/or hyperandrogenemia but have normal ovulation and women who have PCOM and irregular ovulation but no sign of androgen excess.[5]

The AE-PCOS Society criteria (2006) attempted to make a balance between the NIH and Rotterdam definitions and recognized three unique clinical phenotypes (1) Frank PCOS - oligomenorrhea, hyperandrogenism, and PCO (2) Ovulatory PCOS - hyperandrogenism, PCO, and regular menstrual cycles (3) non-PCO PCOS - oligomenorrhea, hyperandrogenism, and normal ovaries. However, all include hyperandrogenism.[6]

Detailed history was taken. General physical examination and complete systemic examination was done. Venous blood sample of women was collected for S. testosterone, and DHEA levels. All the observations were recorded. Women were then classified as PCOS and its phenotypes based on the three above mentioned criteria.

Institutional review board and ethical committee approval was taken prior to the study. Written informed consent was taken of all women. Statistical analysis was done and conclusions drawn. Continuous variables were summarized as mean and standard deviation while nominal/categorical variables were expressed as percentages.

RESULTS

Presentation of PCOS is not homogenous, but it depends on the presence or absence of three elements: hyperandrogenism (HA), menstrual irregularity (OD), and PCO morphology on ultrasonography (PCO) which makes up the phenotypic classification. Different phenotypes present differently concerning their clinical, metabolic, hormonal profile. These differences suggest that each phenotype of PCOS is a variation of a common syndrome.

The mean age of the women included in the study was 23.72 years, 44.54% were educated more than secondary level. All women belonged to middle and lower socioeconomic class. In the study, most women were from the urban area (64.65%) as the hospital is in the center of the city and is in easy reach of the population.

The diagnosis of PCOS by Rotterdam criteria was 62.85%. By NIH it was labelled in only 22.85%. By the criteria set by AE-PCOS Society, it was diagnosed in 28.57%.

In our study, using the Rotterdam criteria, phenotype D (OD+PCO) was the most prevalent phenotype with 64.5% women belonging to it, followed by phenotype A(HA+OD+PCO) with 21.8%, phenotype C(HA+PCO) with 7.2% and phenotype B(HA+OD) with 6.3% (Table 1).

Majority of women with PCOS belonged to the age group of 22-26 years (50.9%), with the mean age being 23.1 years. The mean age in the various phenotypes was similar; differences in age groups across the phenotypes was not found to be statistically significant (p>0.05).

By the NIH criteria, only 16 women (22.8%) were diagnosed as PCOS, of which 50% had hyperandrogenemia and ovulatory dysfunction (Table 1). By the AES criteria,20 women (28.5% were labelled as PCOS,14 (20 %) of which had frank PCOS (Table 1).

Table 1: Diagnosis of PCOS and its phenotypes according to different criteria.

Criteria	No.	%
Rotterdam	**44**	**62.8**
Phenotype A	14	20
Phenotype B	4	5.7
Phenotype C	2	2.85
Phenotype D	24	34.28
NIH	**16**	**22.8**
HA+HI+OD	4	5.7
HI+OD	4	5.7
HA+OD	8	11.4
AE-PCOS society	**20**	**28.5**
Frank PCOS	14	20
Non PCO PCOS	4	5.7
Ovulatory PCOS	2	2.85

The diagnosis by the NIH and AES criteria was significantly lower than that by Rotterdam one, as those without hyperandrogenism were not included under these two.

Levels of dihydrotestosterone sulphate and serum testosterone levels were high in most women diagnosed as PCOS. They were highest in phenotype A (of Rotterdam) than other groups, least being in phenotype D(OD+PCOM) (Table 2).

Table 2: Comparison of androgen level across different phenotypes.

Rotterdam criteria	DHEAS (nmol/l)	Testosterone (nmol/l)
Phenotype A	250.42	3.5+1.2
Phenotype B	195	3.1+1.4
Phenotype C	199	3.3+1.2
Phenotype D	159	2.5+1.1

DISCUSSION

Various hypothesis has been given to explain the heterogeneity in clinical presentation; it could be an interplay between genetic and environmental factors which affect the pathogenesis of PCOS. Another possible explanation given is the intrauterine exposure to maternal androgens which might be responsible for a particular phenotype. It is suggested that excessive androgen exposure to the fetus affects the hypothalamic-pituitary-ovarian axis resulting in adverse reproductive and metabolic outcomes.[7]

In a study done by Sachdeva et al in Chandigarh in 2019, the most common PCOS phenotype was phenotype A which had a prevalence of 67.7%. The prevalence of phenotypes B, C, and D were 11%, 17.7%, and 3.6% respectively, which differs from our findings.[8]

In another study conducted by Gluszak et al in 2012, the prevalence of phenotypes A, B, C, and D were reported to be 60.2%, 16.1%, 18.3%, and 5.4%, respectively in the Polish population.[9]

Similar results were reported by Pehlivanov et al in 2007; percentages of phenotypes A, B, C and D in the Bulgarian population were found to be 58.6%, 11.4%, 10.0% and 20.0%, respectively.[10]

Studies suggest that most hyperandrogenic women with polycystic ovaries tend to have insulin resistance. However, there is conflicting evidence supporting the presence of such features in women with polycystic ovaries and ovulatory dysfunction but without clinical or biochemical signs of hyperandrogenism. Also, whether these women are at increased risk of infertility or metabolic complications is even less well characterized.[11]

The Rotterdam criteria, while representing an advance in recognizing the broad spectrum of presentation of the syndrome and its phenotypic heterogeneity, also increases the risk of over diagnosing women with PCOS while the diagnosis using NIH or AES criteria the diagnosis was low.

Limitations of the study

The study was performed in a single center which is a tertiary care referral center, thus it is not reflective of the whole population.

CONCLUSION

To conclude, it is crucial to establish the diagnostic criteria for PCOS because the long-term consequences of PCOS are still unclear, and early treatment, including infertility management, may play a role in the prevention of metabolic and cardiovascular diseases. Because of the paucity of data on the new phenotype and its long-term implications, the adoption of the Rotterdam 2003 criteria for defining PCOS may be reconsidered. Additional research characterizing the phenotypes and associated morbidities of PCOS is required.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Research Review Board

REFERENCES

1. Hestiantoro A, Hasani KRD, Shadrina A. Body fat percentage is a better marker than body mass index for determining inflammation status in polycystic ovary syndrome. Int J Reprod Biomed (Yazd). 2018;16(10):623-8.
2. NIH Office of Disease Prevention. Evidence-based Methodology Workshop on Polycystic Ovary Syndrome. 2012 Expert Panel Guidelines on PCOS. Available at: https://prevention.nih.gov/docs/programs/pcos/FinalReport.pdf. Accessed on 5th December 2012.
3. Alexiou E, Hatziagelaki E, Pergialiotis V, Chrelias C, Kassanos D, Siristatidis C, et al. Hyperandrogenemia in women with polycystic ovary syndrome: prevalence, characteristics and association with body mass index. Horm Mol Biol Clin Investig. 2017;29(3):105-11.
4. Abinaya S, Siva D, Sabitha R, Achiraman S. An Overview of Hyperandrogenism in PCOS and the Prospective Underlying Factors RJLBPCS. Life Science Informatics Publications. 2019;5(1):179.
5. Ribeiro VB, Kogure GS, Lopes IP. Association of measures of central fat accumulation indices with body fat distribution and metabolic, hormonal, and inflammatory parameters in women with polycystic ovary syndrome. Arch Endocrinol Metab. 2019;63(4):417-26.
6. Thathapudi S, Kodati V, Erukkambattu J, Katragadda A, Addepally U, Hasan Q. Anthropometric and Biochemical Characteristics of Polycystic Ovarian Syndrome in South Indian Women Using AES-2006 Criteria. Int J Endocrinol Metab. 2014;12(1):e12470.

7. 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.
8. Sachdeva G, Gainder S, Suri V, Sachdeva N, Chopra S. Comparison of the Different PCOS Phenotypes Based on Clinical Metabolic, and Hormonal Profile, and their Response to Clomiphene. Indian J Endocrinol Metab. 2019;23(3):326-31.
9. Głuszak O, Stopińska-Głuszak U, Glinicki P, Kapuścińska R, Snochowska H, Zgliczyński W, et al. Phenotype and metabolic disorders in polycystic ovary syndrome. ISRN Endocrinol. 2012;2012:569862.
10. Pehlivanov B, Orbetzova M. Characteristics of different phenotypes of polycystic ovary syndrome in a Bulgarian population. Gynecol Endocrinol. 2007;23(10):604-9.
11. Barth JH, Field HP, Yasmin E, Balen AH. Defining hyperandrogenism in polycystic ovary syndrome: measurement of testosterone and androstenedione by liquid chromatography-tandem mass spectrometry and analysis by receiver operator characteristic plots. Eur J Endocrinol. 2010;162(3):611-5.

Cite this article as: Singh N, Hooja N, Yadav A, Bairwa P, Jaiswal A. Comparison of the various diagnostic criteria used in polycystic ovary syndrome. Int J Reprod Contracept Obstet Gynecol 2022;11:2180-3.