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Genetic correlation by pedigree analysis in patients diagnosed with PCOS as per Rotterdam's criteria

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Abstract
Background: Polycystic Ovary Syndrome (PCOS) is a common endocrine disorder affecting approximately 9.13% of women of reproductive age in South India. It is a major cause of oligomenorrhea, hirsutism, and anovulatory infertility. Despite extensive research, the etiology of PCOS remains elusive. Studies have indicated a hereditary component for various PCOS phenotypes in both female and male offspring, although the precise mode of inheritance remains unclear.
Objectives: To gather evidence supporting a genetic basis for Polycystic Ovary Syndrome (PCOS) by examining phenotypic characteristics in male and female relatives of PCOS patients.
To determine the mode of inheritance of PCOS phenotypes through pedigree analysis.
Methodology: 15 probands were recruited randomly from consenting patients diagnosed with PCOS as per the Rotterdam Criteria. Thorough examination of family history was used to assign affected status to relatives of each proband. Female relatives were assessed for degree of hirsutism using the modified Ferriman Gallwey (mFG) method as well as menstrual irregularities. Females with hirsutism (mFG score >7)\acanthosis\male pattern hair loss along with menstrual irregularities were assigned as expressing PCOS phenotype. Male relatives were assessed for male pattern baldness (MPB) based on Hamilton-Norwood scale and early onset (<30 yrs. Old) MPB with score >4 was considered a PCOS phenotype. Pedigrees were mapped for each family and the pattern of inheritance traced. Segregation analysis was also employed to further ascertain the pattern of inheritance.
Results: Of the 15 pedigrees analysed, eight showed a simple Mendelian pattern of Autosomal Dominant Inheritance, seven showed an autosomal dominant pattern with incomplete penetrance in males.
Conclusion: Based on the thorough examination of family history and the phenotypic presentations of male and female relatives of patients affected by PCOS, evidence was obtained for the genetic basis of polycystic ovary syndrome (PCOS). The mode of inheritance of PCOS phenotypes was found to be consistent with an autosomal dominant pattern of inheritance. These findings suggest that first-degree family relatives of PCOS probands have a significantly higher risk of developing PCOS. Overall, the results of this study provide important insights into the heritability and mode of inheritance of PCOS, which can aid in the development of targeted prevention and treatment strategies for this common female endocrinopathy.

Keywords: PCOS, genetic correlation, pedigree analysis, autosomal dominant inheritance, hirsutism

Introduction
Polycystic Ovary Syndrome (PCOS) also known as Stein Leventhal syndrome is a commonly reported female endocrinopathy affecting women in the reproductive age group. Recent researches report the prevalence of PCOS in South India as 9.13% [16]. It remains the most common cause of oligomenorrhea, hirsutism and anovulatory infertility. These patients possess a higher risk of developing infertility, dysfunctional uterine bleeding, endometrial carcinoma and several metabolic disorders including Insulin resistance, hyperinsulinemia and diabetes mellitus, hypertension and dyslipidemia [14, 21, 24]. The aetiology of this syndrome remains largely unknown, but mounting evidence suggests that PCOS might be a complex multigenic disorder with strong epigenetic and environmental influences [3, 9]. Studies showing familial clustering of PCOS cases and greater concordance of symptoms of PCOS in identical twins and the heritability of endocrine and metabolic features of PCOS all strongly suggest the involvement of genetic mechanisms [7, 13]. Even though the search for candidate genes in PCOS has yielded some positive results, the controversy on the mode of inheritance (e.g. Autosomal dominance, autosomal recessive, X-linked, polygenic, oligogenic) persists [19]. It is clear that genetic factors play an important role in the development of PCOS, but studies on this view in India are scanty.

This background knowledge demands the necessity to work out the genetic basis of PCOS and this study is aimed to provide evidence for the same by working out the association between a family history of phenotypes suggestive of PCOS and the incidence of PCOS. Epidemiological studies have shown that PCOS can occur in men as well, be it with a different phenotype than in females, but most of the studies analysing the genetic basis of PCOS are solely focused on female relatives of the proband. Premature male pattern balding before the age of thirty is now established as a symptom of the male PCOS equivalent [5, 11, 15]. In this study, women diagnosed with PCOS were interviewed and their family history was examined by drawing pedigrees and mapping the expressions of various PCOS phenotypes in males and females. We intended to measure the prevalence of these phenotypes in the blood relatives of the proband to test the strength of association between the familial expression of these phenotypes and developing PCOS and to determine the pattern of inheritance of the phenotype. This would provide evidence of a genetic component for PCOS without the use of any invasive investigations. A clear understanding of the aetiology of PCOS is important for the development of effective screening and novel therapeutic and preventive strategies. Our findings also assist the general public in analysing the risk of developing PCOS and provides an opportunity for early interventions through lifestyle modifications.

Review of literature

Polycystic ovary syndrome was first described by Stein and Leventhal as a chronic disorder with unknown aetiology in 1935. A genetic basis for PCOS was suggested for the first time about 50 years ago by Cooper *et al* in 1968 [7]. Family history of PCOS is a known risk factor for PCOS. Numerous studies have been conducted to understand the mode of inheritance of this syndrome where presence of PCO on ultrasound is accepted as the female phenotype, and premature balding has been suggested as the male counterpart [15, 20]. These studies have suggested that PCOS follows a simple Mendelian pattern of inheritance. Cooper *et al*. suggested a dominant mode of inheritance in Caucasian first-degree relatives. Carey *et al*. determined the mode of inheritance for 10 large families taking premature male pattern baldness (PMPB) as the male phenotype, segregation analysis showed an autosomal dominant inheritance, consistent with a single gene defect [5]. This was confirmed by Govind *et al*. in a study that compared 29 PCOS probands with 10 controls [1]. However, a twin study did not confirm PCOS to be an autosomal genetic disorder [23]. Thus most of the existing literature seems to suggest an autosomal dominant mode of inheritance for PCOS.

Nearly 70 years after its discovery, the criteria that is used presently globally to diagnose PCOS was formulated by the Rotterdam consensus. Polycystic ovarian syndrome (PCOS) is defined by the presence of two of three of the following criteria: oligo-anovulation, hyperandrogenism and polycystic ovaries. [18].

The clinical presentation of the syndrome is variable. Oligo-anovulation can present as oligomenorrhea, amenorrhea, or prolonged erratic bleeding [12]. Hirsutism is the major clinical presentation of hyperandrogenism occurring in women with PCOS [2, 22]. It is evaluated using the modified Ferriman-Gallwey scoring system [10]. This tool is used to evaluate hair growth at nine sites: upper lip, chin/face, chest, upper and lower back, abdomen, pubic hair, arms, and thighs. A score of 0 is given in the absence of terminal hair growth and a score of 4 is given for extensive growth. A total score of 8 or more is indicative of hirsutism. The Rotterdam consensus group defined the presence of 12 or more follicles measuring between 2 and 9 mm in diameter and/or an increased ovarian volume of greater than 10 cm3 as the criteria for polycystic ovarian morphology. This presentation in one ovary is sufficient for the diagnosis of polycystic ovary [18]. We have relied on this criteria to select our study participants.

Aims and Objectives

The primary aim of this study was to determine how the maternal or paternal phenotype correlate to the diagnosis of PCOS as per Rotterdam's criteria by drawing and analysing pedigree charts using premature balding in males and PCOS/anovulatory cycles/male pattern balding/hirsutism in females as the phenotypic parameters.

Through this correlation we aimed to provide evidence for the genetic basis of PCOS.

We also aimed to map the pattern of inheritance of Polycystic Ovarian Syndrome (PCOS) in the selected families by the analysis of pedigree charts.

Materials and Methods

The study was conducted in a clinical setting, assessing the patients diagnosed with PCOS presented to the gynaecology OPD within the reproductive age group of 14 - 45 years. Ethical approval for this study was obtained from IEC (IEC Study Ref No: 22/20/IEC/JMMC&RI). The diagnosis of PCOS was according to the Rotterdam European Society of Human Reproductive Embryology PCOS group's revised criteria with presence of 2 out the 3 criteria. The criteria are, 1]. Oligo and/or anovulation 2]. Clinical and/or biochemical signs of hyperandrogenism 3]. Polycystic ovaries. Oligo and/or anovulation was defined as menstrual cycles more than 45 days in length or less than 9 cycles per year [18]. Clinical signs of hyperandrogenism were defined as presence of hirsutism, persistent acne (not reacting to dermatologic treatment) or androgenic alopecia. Hirsutism was defined as a modified Ferriman-Gallwey score of >8. Polycystic ovaries (PCO) were diagnosed on pelvic USG by presence of ≥12 follicles measuring 2-9 mm in diameter and/or ≥10 ml ovarian volume [4].

Study Participants

Study participants were selected at random from patients visiting Gynaecology OPD and enquired about family history. Since it's a pilot study from Southern India and takes into account the entire blood relation of the proband, 15 families with sufficient members for further study were randomly selected. Inclusion criteria included the following (1) Diagnosis of PCOS as per Rotterdam criteria (2) Proband should have at least one sibling and (3) exclusion of other disorders, such as Cushing syndrome, hyperprolactinemia, and hypothyroidism. Hyperprolactinemia and hypothyroidism were excluded by normal levels of PRL and TSH, respectively; Cushing syndrome was excluded by clinical evaluation.

Informed consent was obtained from all of the participants who were provided with a detailed information sheet. Health status, general medical history, medication, diagnostic tests, and other relevant individual information were taken into account. All female family members had their degree of hirsutism assessed using the modified Ferriman and Gallwey score [10]. A score above 8 was considered as PCOS phenotype for this study. A careful reproductive history was taken for each woman and any menstrual disturbances were duly noted. Other symptoms of

PCOS, namely, acanthosis, male pattern baldness and excessive acne were also enquired about and noted. Female relatives with any of the aforementioned symptoms in addition to menstrual irregularities were assigned as expressing PCOS phenotype. Each male family member was assessed for the degree and time of onset of balding based on the Hamilton-Norwood scale. Male pattern hair loss at stage 4 before the age of 30 was considered a PCOS phenotype in this study. The selected fifteen families of PCOS probands completed the study by screening all blood relatives up to their second super generation. All the probands were of South Indian origin. Forty-eight first-degree relatives were identified from the fifteen PCOS probands (Table 1). 8 brothers were below the age of 30 and therefore unsuitable for status assignment as they may become bald before the age of 30.

Study Design

The study followed the pattern of a retrospective cross-sectional. A group of individuals diagnosed with the pathology (PCOS) were observed to check for the expression of a factor (here the factor being phenotypic expression of PCOS in paternal and maternal family members) to determine the factor's influence on incidence of PCOS. The factors chosen were Premature balding in male relatives and Hirsutism/Anovulatory cycles/PCOS in female relatives. The factors were chosen based on various studies establishing premature balding as the male equivalent of PCOS [5, 11, 15]. The sample size was taken as 15 since it is a pilot study that takes into account the pedigree of the entire blood relation of the subject with PCOS. The recruited patients took part in a survey answering a symptom questionnaire about each of their blood relatives. Females with conditions other than PCOS causing hirsutism/hyperandrogenism such as adrenal hyperplasia, Cushing syndrome, androgen therapy or use of the drug Danazol were considered unaffected. Data collection tools and techniques included a thorough clinical examination of the presented patient and answering a symptom questionnaire to assess the history of PCOS symptoms in female relatives and premature baldness in male relatives in their corresponding generation, immediate super generation, and second super generation. A pedigree chart was drawn for each family depicting at least 3 generations. The genetic correlation if present was assessed and the pattern of inheritance traced for each pedigree.

Table 1: Distribution of first-degree relatives of PCOS probands and assignment of PCOS phenotype

	Probands	Mothers	Fathers	Sisters	Brothers	Total first degree relatives
Included in the study	15	15	15	5	13	48
Total females	15	15		5		20
Females assigned with the PCOS phenotype		5		2		7
Normal females		10		3		13
Unassigned females		0		0		0
Total males			15		13	28
Males with premature male pattern balding			5		3	8
Normal males			10		2	12
Unassigned males					8	8

Pedigree Analysis

In this study, 15 pedigrees were mapped out from the family history provided by the study participants. We examined each of the 15 pedigrees separately to identify the pattern of inheritance. We also applied segregation analysis to calculate the segregation ratio for the first-degree relatives with PCOS phenotype on the assumption of an autosomal dominant pattern of inheritance based on previous similar studies.

Data analysis plan: Univariate, bivariate and multivariate analysis including ANOVA.

Data analysis: IBM SPSS version 23

Observations and Results

The mean age of the 15 PCOS subjects examined was 25.47±10.53 years. Table 2 shows the number of first and second generation family members assessed for each subject and the pattern of inheritance traced by pedigree analysis. Of the 10 PCOS proband families, 5 of 15 mothers (33.3%), 5 of 15 fathers (33.3%), 2 of 5 sisters (40%), and 3 of 13 brothers (23%) [3 of 5 assignable brothers (60%)]. expressed PCOS phenotype. First-degree female relatives of affected individuals had a 35% chance of being affected. Of the first-degree male relatives, 28.5% [and 40% of assignable male relatives]. were found to have phenotype suggestive of PCOS. Of the 15 proband families, there were 8 families with at least 1 affected parent. The transmission of PCOS phenotype did not show any variation between maternal and paternal side of the family, i.e.; PCOS phenotypes were transmitted equally on both maternal and paternal side of the family.

Of the 15 pedigrees analysed, 8 showed a simple Mendelian pattern of Autosomal Dominant Inheritance (P02, P03, P05, P10, P11, P12, P13, P14), while 7 showed possible Autosomal dominant pattern with incomplete penetrance in males(P01, P04, P06, P07, P08, P09, P15). 12 out of the total of 439 family members assessed could not be assigned any status as 4 members had passed away before reaching puberty and 8 male relatives were below the age of 30 (and hence could not be ruled out as negative for premature balding). Out of the assignable 427 members, 70 were assigned affected status. Thus, the incidence of PCOS phenotypes among blood relatives of PCOS probands was found to be 16.39% which is significantly higher than the prevalence in the general public (9.13%).

Table 2: Summary of relatives assessed, number affected, and inheritance pattern for each PCOS proband

Subject Id	Age of proband	Number of blood relatives assessed	Number of relatives affected	Pattern of inheritance
1	24	39	6	Autosomal Dominant Incomplete Penetrance
2	24	22 *	2	Autosomal Dominant
3	22	32	9	Autosomal Dominant
4	28	28	1	Autosomal Dominant Incomplete Penetrance
5	15	19	7	Autosomal Dominant
6	32	26	4	Autosomal Dominant Incomplete Penetrance
7	27	29	3	Autosomal Dominant Incomplete Penetrance
8	36	35	7	Autosomal Dominant Incomplete Penetrance
9	25	32	2	Autosomal Dominant Incomplete Penetrance
10	28	13	1	Autosomal Dominant
11	24	28	11	Autosomal Dominant
12	21	37 #	11	Autosomal Dominant
13	26	24	3	Autosomal Dominant
14	27	36	2	Autosomal Dominant
15	23	39	1	Autosomal Dominant Incomplete Penetrance
*1 Passed away before reaching puberty		#2 Passed away before reaching puberty		

Segregation Analysis

The segregation ratio was calculated considering a total of 10 post pubertal siblings of PCOS probands, 5 were assigned affected status, which is exactly the expected value of 50% for an autosomal dominant pattern of inheritance (Table 3). Considering the first degree relatives, 15 were assigned affected status and 25 were assigned as unaffected, while 8 could not be assigned any status. Assuming an autosomal dominant pattern, the expected value is 20, giving a segregation ratio of 15/20 (χ-square, 0.252) Pearson's chi-square test [17]. was employed taking a P value of 0.05 as the cut off level and the computed χ-square analysis showed that there is no significant deviation of the observed value from the expected when assuming an autosomal dominant mode of inheritance.

Table 3: Segregation analysis of PCOS phenotype among siblings and first-degree relatives

N = 15	Number	Observed	Expected	Ratio (O/E)	Chi square
Siblings	18				
Assignable	10				
Affected status	5	5	5	1:1	
First degree relatives	48				
Assignable	40				
Affected status	15	15	20	3:4	0.252

Discussion

Analysis of family pedigrees of PCOS patients has clearly demonstrated that family history of PCOS phenotypes is a predisposing factor for developing the syndrome proving the existence of a genetic component. Both the maternal and paternal side of the family seem to transmit the phenotype of the syndrome equally. The probability of developing PCOS is twice increased if an individual has an affected first or second generation family member as the prevalence of PCOS among family members of PCOS probands was found to be 16.39% which is nearly 2 times greater than prevalence in the general population.

Even though the search for a single gene or pathway involved in developing PCOS has been largely futile, the familial clustering of this syndrome indicates a genetic basis for the etiopathogenesis of PCOS [13]. The pattern of heredity too has remained elusive despite many genetic studies. Yet most of the family studies seem to agree on a simple Mendelian pattern of inheritance consistent with either an autosomal dominant or X-linked pattern of inheritance. Pedigree analysis of 15 families selected in this study all showed an autosomal dominant pattern. The study conducted by Govind *et al* concluded that polycystic ovaries were inherited as an autosomal dominant trait by comparing the segregation analysis done on affected first degree family members of 29 PCOS cases and 10 unaffected controls. The percentage of affected first degree relatives (52% of the mothers, 21% of the fathers, 66% of the sisters, 22% of brothers) suggested an autosomal dominant inheritance caused by the same gene. Similarly in all the cases analysed here, segregation ratio of observed: expected was consistent with an autosomal dominant inheritance. The seven pedigrees that deviated from this result could still be considered as showing an autosomal dominant pattern if we consider incomplete penetrance in males. These discrepancies could be explained by the fact that genetic influence alone does not drive the pathogenesis of any disorder. External factors including differences in races and geographical area included in these studies, and individual differences due to diet and lifestyle factors could be behind these variations in results.

Limitations and Implications

This study was conducted as a survey and did not involve blood tests, so it does not provide information about the biochemical aspects of PCOS. However, when combined with genetic studies on PCOS that use blood serum analyses, this study helps to establish a genetic link to a common disorder that can be understood and assessed by the general public without the need for invasive tests.

Better understanding of the mode of inheritance will assist diagnosticians in stipulating the right approach to a diagnosis through family history. It is worth noting that metabolic syndrome linked with PCOS is a preventable disorder through non-medical interventions, underscoring the importance of identifying the target population for implementing effective prevention strategies. Furthermore, this study provides valuable insights for future research on PCOS, as molecular genetic approaches can be employed to assess the contributions of individual genes when clear phenotypic markers and their mode of transmission across generations are known.

Conclusion

Pedigree analysis of 15 probands showed a clear picture of Autosomal Dominance with a probable incomplete penetrance in males. First-degree family relatives of PCOS probands were found to have a significantly higher risk of developing PCOS

symptoms. Understanding the heritability of PCOS, independent of attempts to detect specific molecular abnormalities present is of great importance. Studies of the heritability of PCOS will assist in confirming the mode of transmission. Studies that define the familial risk of the disorder are critical for counselling families with affected individuals. Tracing a pattern of inheritance solely based on phenotypic characteristics not requiring invasive investigations will help the general public in predicting the risk of PCOS. Timely therapeutic and lifestyle interventions aimed at the high-risk population will improve the management of PCOS during adolescence, prevent associated comorbidities and improve quality of life.

Summary

This study aimed to obtain evidence for the genetic basis of polycystic ovary syndrome (PCOS) by screening blood relatives of women affected by PCOS and to determine the mode of inheritance of PCOS phenotypes through pedigree analysis. The probands were recruited randomly from patients presenting to a gynaecology OPD, diagnosed with PCOS as per the Rotterdam Criteria. The families of 15 probands consented to participation in the study. A thorough examination of family history was used to assign affected status in male and female relatives. Female relatives were assessed for degree of hirsutism using the modified Ferriman Gallwey (mFG) method as well as menstrual irregularities. Females with hirsutism (mFG score >7)\acanthosis\male pattern hair loss along with menstrual irregularities were assigned as expressing PCOS phenotype. Each male family member was assessed for the degree and time of onset of male pattern baldness (MPB) based on the Hamilton-Norwood scale and early onset (<30 yr old) MPB with a score >4 was considered a PCOS phenotype. All relatives were assigned affected or unaffected status based on the presence or absence of the aforementioned PCOS phenotypes, pedigrees were mapped for each family and the pattern of inheritance was traced. Segregation analysis was also employed to ascertain the pattern of inheritance.

Of the 15 pedigrees analysed, eight showed a simple Mendelian pattern of Autosomal Dominant Inheritance and seven showed possible autosomal dominant pattern with incomplete penetrance in males. Thus, the inheritance of PCOS phenotypes was found to be consistent with an autosomal dominant pattern of inheritance. This is in agreement with existing literature. Of the 10 PCOS proband families, 5 of 15 mothers (33.3%), 5 of 15 fathers (33.3%), 2 of 5 sisters (40%), and 3 of 13 brothers (23%) [3 of 5 assignable brothers (60%)]. were assigned affected status. First-degree female relatives of affected individuals had a 35% chance of being affected, which is greater than the 9.15% probability of the general public. Of the first-degree male relatives, 28.5% [and 40% of assignable male relatives]. were found to be affected.

Out of the ten assignable siblings, 5 were affected as expected for an autosomal dominant trait. Out of the 40 assignable first-degree relatives, 15 were affected, producing a segregation ratio of 15/20, which is still consistent with the autosomal dominant pattern of inheritance ($\chi 2 = 0.252$).

Collectively, this data indicates that genetic mechanisms play at least some role in the pathogenesis of PCOS.

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Declarations

Ethical Clearance

This study was conducted after obtaining ethical clearance from the board of Institutional Ethical Committee of Jubilee Mission Medical College and Research Institute, Thrissur.

Consent

Participation in this study was on a voluntary basis from the population satisfying inclusion criteria. All participants of this study were included in the study after giving informed consent in writing. All identifying information in the data collected was removed and replaced with randomly allotted numbers before segregation analysis.

Author's contributions

Dr S.D. formulated the research question and outlined the study methodology, analysed and interpreted the patient data regarding the diagnosis of PCOS and attributed affected and non-affected status based on phenotypes. Dr A.H.M contributed to formulating the study design, collected and segregated data and plotted pedigrees, assisted in segregation analysis and was a major contributor in writing the manuscript. Dr S.R. contributed to formulating the study design, interpretation of pedigrees and segregation analysis in determining the mode of inheritance. All authors read and approved the final manuscript.

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