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Frequency of Polycystic Ovary Syndrome among the Students of a Medical College in Dhaka City

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Abstract:

Background: Polycystic ovary syndrome (PCOS) is a common heterogeneous endocrine disorder. Only few data are available in Bangladesh. The aim of this study was to find out the frequency of PCOS among the students of a medical college in Dhaka city. Materials & Methods: This cross sectional study was done among the 3rd to 5thyear students of Ibrahim Medical College. We used Rotterdam criteria (Revised 2003) for diagnosis of PCOS. After taking written consent a detailed history including menstrual history was taken. Anthropometric measurements including height in cm, weight in kg, waist circumference (WC) were measured. We assessed hyperandrogenism (H) clinically by hirsutism using a modified Ferriman-Gallway(mFG) method. The presence of acne and acanthosisnigricans was also noted. For biochemical hyperandrogenism we did total testosterone estimation. Blood samples was collected between 08.00 and 10.00 am on Days 2-7 of a spontaneous bleeding episode or randomly in the case of amenorrhea after an overnight fast. The circulating levels of total testosterone, levels were measured by Chemiluminescent Immunoassay (Advia Centaur XP^{TM}). Transabdominal USG (Aloka F37) was done by expert radiologist of department of Radiology, BIRDEM. PCOS group was categorized in 4 different phenotypes based on the presence of oligo-anovulation (O), hyperandrogenism (H) and polycystic ovarian morphology (P): (i) Phenotype A (O+ H+ P), (ii) Phenotype B (O+H), (iii) Phenotype C (H+P) and (iv) Phenotype D (O+P).Results: Out of 73 girls 27(37%) satisfied Rotterdam's criteria for PCOS. Phenotype B was more common 16(59.3%) followed by A 5(18.5%) then C 3(11.1%) and D 3(11.1%). Hirsutism was found in 24 (88.8%) girls, menstrual disturbance was also present among 24 (88.8%) girls, serum testosterone was elevated in 6(22.2%) girls, 11(40.7%) had USG findings of polycystic ovaries. The mean BMI (24.70 $\pm$ 3.7) was significantly high (BMI $\geq$ 23 kg/ m^2) among those who had PCOS (59.86%). Acne &acanthosisnigricans was observed 15(55.6%) & 6(22.2%) girls among PCOS respectively which were not statistically significant. Hypothyroidism was observed 7(25.9%) girls with PCOS. Associated family history of DM found in 21(77.8%) girls with PCOS. Family history of PCOS was present in 7(25.9%) girls in PCOS group. The difference were statistically significant ($p<0.05$) between two groups. Conclusion: Prevalence of PCOS is 37% which demonstrates that PCOS is an emerging disorder. This draws attention to the issue of early diagnosis which could provide opportunity to target the group to prevent future morbidities.

Key words:PCOS, Hyperandrogenism, Hirsutism, Acanthosisnigricans, Oligo-anovulation

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I. Introduction:

Polycystic ovary syndrome (PCOS) is the most common endocrine disorder amongst women of reproductive age. It is a heterogenous disorder of uncertain etiology, but there is strong evidence that complex interactions between genetic, environmental and behavioral factors contribute to causing this syndrome. Incidence of PCOS in women of reproductive age is reported to be 5 to 10% which is found increasing than assumed. PCOS is characterized by anovulatory menstrual cycle, infertility, hyperandrogenism and clinically manifested by irregular menstruation, hirsutism, obesity & acne thus resemble different metabolic syndromes[1]. In the last two decades, three alternative definitions have been formulated for the diagnosis of PCOS. The most widely used 1990 National Institutes of Health (NIH) criteria[2] include clinical and/or biochemical hyperandrogenism and chronic anovulation. The 2004 Rotterdam criteria (Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long term health risks related to polycystic ovary syndrome) suggest PCOS should be diagnosed by two of the following three criteria: oligo-anovulation, clinical or biochemical hyperandrogenism and poly-cystic ovaries (PCOs) on ultrasound. The most recent Androgen Excess and PCOS Society (AE-PCOS Society) criteria[3] recommend that PCOS should be defined as clinical or biochemical hyperandrogenism associated with ovulatory dysfunction (OD) in the form of oligo-anovulation or PCOS. All three sets of criteria highlight exclusion of other related disorders before making a diagnosis of PCOS[3].

The different diagnostic criteria create several phenotypes of PCOS. For simplification, the phenotypes were divided into four diagnostic groups: phenotype A (NIH PCOS of biochemical/clinical hyperandrogenism and oligo/anovulation with PCO); phenotype B (NIH PCOS of biochemical/clinical hyperandrogenism and oligo/anovulation without PCO); Phenotype C (non-NIH PCOS with biochemical/clinical hyperandrogenism and PCO but with normal ovulation); phenotype D (non-NIH PCOS with oligo/anovulation and PCO but without any biochemical/clinical hyperandrogenism)[4]. Also in the literature we find differences in prevalence percentages for NIH and Rotterdam criterion. One study reported a prevalence of 26% according to Rotterdam criteria and 8% according to NIH criteria[5]. Similar reports were observed in a recent survey on 728 women wherein the prevalence was 8.7% according to NIH criteria and 17.8% according to Rotterdam criteria.[6]

The prevalence seems to vary widely in different countries. In the U.S., two studies that used NIH criteria have documented prevalence rates of 4% in a population of 400 womenand 6.6% in women from a southeastern university[7]. Prevalence among women from other races appears to be similar. A study on 154 Caucasian women in Madrid, Spain, also found similar prevalence rates of 6.5%.[8]Thus, it appears that the prevalence of clinically evident PCOS in women of reproductive age in Europe and America ranges from 6.5 to 8.0% using the 1990 NIH criteria and it rises two- to three-fold if the Rotterdam criteria are applied.

The prevalence in Asian countries appears to be lower, with a reported prevalence of 2.4% in China[9] and 6.3% in Sri Lanka[10](Rotterdam criteria).Women with PCOS may present with multiple manifestation which include cutaneous, reproductive and metabolic abnormalities. Cutaneous manifestations include hirsutism; acne and male pattern of baldness are caused by hyperandrogenism. Reproductive manifestations include menstrual dysfunctions (secondary amenorrhea, oligomenorrhea, infertility, early pregnancy loss and other complication of pregnancy). Metabolic and endocrine manifestation include increased circulating levels of total &/or free testosterone, androstenedione, dihydroepiandrostenedione sulfate (DHEAS); decreased sex hormone binding globulin (SHBG); increased insulin level and increased LH/FSH ratio[11].These women have an increased risk of insulin resistance and hyperinsulinemia, an increased risk of glucose intolerance and type 2 diabetes mellitus, dyslipidemia, subclinical atherosclerosis, and vascular dysfunction, independent of body mass index (BMI)[12].Women with PCOS have multiple risk factors for diabetes including obesity, a family history of type 2 diabetes, and abnormalities in insulin action (both insulin resistance and beta-cell dysfunction). There is now clear evidence that women with PCOS are at increased (3-7 times) risk of developing type 2 diabetes[3].

The prevalence of insulin resistance (IR) in PCOS patients ranges from 44 to 70%. This wide range may be due to several factors, including the heterogeneity of the diagnostic criteria for PCOS employed in these studies, the genetic background among the assessed population (Shaw et al. 2008) and differences in the methods used for defining IR. The presence of chronic anovulation associated with higher androgen levels correlates with lower insulin sensitivity and higher prevalence of cardiovascular risk factors, such as IR, impaired glucose tolerance (IGT), T2DM, dyslipidemia and metabolic syndrome (MetS). However, the presence of two PCOS phenotypes identified according to the Rotterdam criteria-hyperandrogenism and polycystic ovaries with ovulatory cycles and anovulation and polycystic ovaries without hyperandrogenism show little or no evidence of IR using surrogate markers[13].

II. Methods and Materials

This cross sectional study was conducted in the Ibrahim Medical College from April 2019 to September 2019 with sample size 73. Initially we arranged an interactive introductory lecture where the study design & purpose was described to the students. They were asked to send SMS via mobile who are interested to participate in our study. After getting their reply, they were asked to report to us. Then we re-explain the study procedure & informed written consent had taken. Subject selection was done as per inclusion and exclusion criteriawere subjects having congenital adrenal hyperplasia, Cushing's syndrome, Women on medication like oral contraceptives, glucocorticoids, Refusing to give written consent. A detailed history was obtained. Data was collected in a structured questionnaire. Anthropometric measurements including height in cm (bare foot, standing erect against wall), weight in kg (in a weighting scale, bare foot, light clothing), waist circumference (WC) were measured by placing a tape horizontally midway between the lower border of the ribs and iliac crest on the mid-axillary line and hip circumference (HC) wasmeasured to the nearest centimeter at the greatest protrusion of the buttocks. The BMI was calculated by dividing the weight (in kg) by the height (in m) squared to assess obesity. Systolic and diastolic blood pressure was measured twice in with 1-2 hour gap in one visit with a mercury sphygmomanometer. The amount of terminal hair growth was assessed using a modified Ferriman-Gallway method (Ferriman and Gallway, 1961) in which the upper lip, chin, chest, upper and lower abdomen, thighs, upper and lower back and upper arms were scored from 0 to 4. We used an mF-G score of 6 as the upper normal limit in accordance with a study in South Asia. The girl were asked to compare the amount of body hair they had with a picture displaying the degree of hair growth in nine regions. Hirsutism scores recorded by the girls were checked for accuracy during clinical examination by the researcher & corrected if necessary. The presence of acne and acanthosisnigricanswas noted. For testosterone assay students were requested to come between 08.00 and 10.00 am on Days 2-7 of a spontaneous bleeding episode or randomly in the case of amenorrhea after an overnight fast. Blood sample (2ml) was taken in the Endocrine Laboratory of BIRDEM. Serum was separated by centrifugation & stored at -20^0 c until it was analyzed. The circulating levels of total testosterone, levels were measured by Fully Automated Bidirectionally Interfaced ChemiluminescentImmunoassay with sensitivity of 10 ng/dl (ByAdvia Centaur XPTM by Testosterone Kit) &transabdominal USG (By Hitachi Aloka F37) was done by postgraduate radiologist of department of Radiology, BIRDEM.Data was analyzed by computer with the help of SPSS 22.0 (Statistical Package for Social Science). Statistical analyses was done by using appropriate statistical tool like 'chi-square' test, student's't' test, where applicable. Statistical significance is set at 0.05 level and confidence interval at 95% level. The study was conducted after getting clearance from Ethical review committee, Bangladesh Diabetic Society.

III. Results:

Figure I: Distribution of PCOS among study population (n=73)

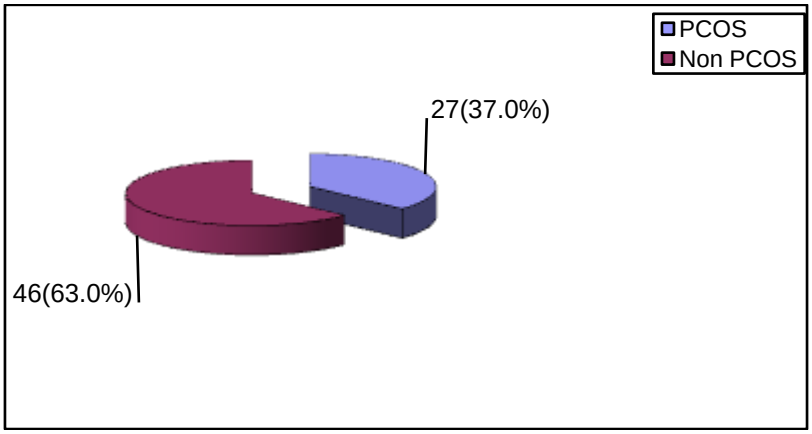

Figure I shows that out of 73 girls, 27 (37%) has fulfilled PCOS diagnosis according to Rotterdam criteria

Table I Distribution of the study population by clinical & laboratory parameter (n=73)

Clinical parameter	PCOS (n=27)		Non PCOS (n=46)		P value
	n	%	N	%	
Hirsuitism (mFG score>6)	24	88.9	8	17.4	[a]0.001[s]
Acne	15	55.6	19	41.3	[a]0.239 [ns]
Oligomenorrhoea	23	85.1	9	19.6	0.001[s]
Amenorrhoea	1	3.7	0	0	0.189[ns]
Acanthosisnigricans	6	22.2	9	19.6	0.746[ns]
BMI (Mean±SD)	24.70±3.7		23.03±3.28		[b]0.049s
Waist/Hip ratio	0.8±0.05		0.78±0.05		[b]0.103[ns]
HighTestosterone>.91ng/ml	6	22.2	0	0.0	0.001[s]
Polycystic Ovaries on USG	11	40.7	1	2.2	[a]0.001[s]

[i]s= significant, [a]p value reached from chi square test, [b]p value reached from unpaired t-test
Table I shows Hirsutism was found in 24 (88.8%) girls, menstrual disturbance was also present among 24 (88.8%) girls, serum testosterone was elevated in 6(22.2%) girls, 11(40.7%) had USG findings of polycystic ovaries. The mean BMI (24.70 $\pm$ 3.7) was significantly high among those who had PCOS

Table II: Features of PCO on USG in study population(n=73)

PCO ovaries on USG	PCOS (n=27)		Non PCOS (n=46)		P value
	n	%	n	%	
Presence of both >12 cysts(2-9 mm) & increased ovarian volume >10cm^3	8	29.6	0	0.0	0.001[s]
Ovarian volume >10cm^3	9	33.3	1	2.2	0.001[s]
>12 cysts(2-8 mm)	9	33.3	1	2.2	0.001[s]

s= significant, p value reached from chi square test
Table II shows, among 11 (40.7%) polycystic ovaries, 29.6% had both the features of polycytic ovaries, 33% has increased ovarian volume & typical cystic pattern was found in 33% of PCOS group

Table III: Presence of other illness among study population (n=73)

History of other illness	PCOS (n=27)		Non PCOS (n=46)		P value
	n	%	N	%	
Hypothyroidism	7	25.9	3	6.5	0.020[s]

Hypertension	1	3.7	0	0.0	0.189[ns]

s= significant, ns= not significant, p value reached from chi square test
Table III shows that hypothyroidism was observed in 7 (25.9%) girls among the PCOS group

Table IV: Family history of study population (n=73)

Associated family history	PCOS (n=27)		Non PCOS (n=46)		P value
	n	%	n	%	
DM	21	77.8	24	52.2	0.030[s]
HTN	12	44.4	17	37.0	0.528[ns]
PCOS	7	25.9	2	4.3	0.007[s]
Obesity	2	7.4	2	4.3	0.579[ns]

s= significant, ns= not significant, p value reached from chi square test
Table IV shows, associated family history of DM found in 21(77.8%) girls with PCOS. Family history of PCOS was present in 7(25.9%) girls in PCOS group.

Table V: Distribution of the study subjects by phenotype (n=27)

Phenotype	Number of patients	Percentage
A	5	18.5
B	16	59.3
C	3	11.1
D	3	11.1

Table V shows phenotype of the study subjects, it was observed that majority patients had phenotype B (59.3%), followed by A (18.5%) and then C(11.1%) and D(11.1%).

Table VI: clinical & laboratory parameter of different phenotype of PCOS (n=27)

	Phenotype								P value	Post hoc analysis p value
	A (n=5)		B (n=16)		C (n=3)		D (n=3)			
	n	%	n	%	n	%	n	%		
Age (in years)Mean±SD	22.2±0.8		22.0±1.0		22.9±1.1		23.0±1.0		[a]0.360[ns]	0.989
Hirsutism	5	100.0	16	100.0	3	100.0	0	0.0	[b]0.001[s]	
Menstrual disturbance	5	100.0	16	100.0	0	0.0	3	100.0	[b]0.001[s]	
Acne	4	80.0	7	43.8	2	66.7	2	66.7	[b]0.491[ns]	
Acanthosisnigricans	2	40.0	3	18.8	0	0.0	1	33.3	[b]0.553[ns]	
BMI (kg/m2)Mean±SD	26.9±2.6		23.9±2.1		22.8±1.9		23.4±1.5		[a]0.037[s]	0.03
Waist/Hip ratio Mean±SD	0.8±0.04		0.8±0.02		0.8 ±0.03		0.8±0.02		[a]1.000[ns]	0.872
Elevated Testosterone	1	20.0	5	31.2	0	0.0	0	0.0	[b]0.001[s]	
Polycystic ovaries on USG	5	100.0	0	0.0	3	0.0	3	100.0	[b]0.001[s]	

s= significant, ns= not significant, [a]p value reached from ANOVA test and post hoc analysis done between the group, [b]p value reached from chi square test

Table VI shows that significant difference of demographic, anthropometric, biochemical & USG features among the four different phenotype

Table VII: Association between obesity & different phenotype of PCOS (n=27)

	Phenotype								P value
	A (n=5)		B (n=3)		C (n=16)		D (n=3)		
	n	%	n	%	N	%	n	%	
BMI (kg/m^2)									
<18.5 (underweight)	0	0.0	0.0	0.0	0.0	0.0	0	0.0	
Normal (18.5-22.9)	2	40.0	6	37.4	1	33.3	2	66.7	
Over weight (23.0-26.9)	2	40.0	7	43.8	2	66.7	1	33.3	0.001^s
Obese I (27-29.9)	1	20.0	3	18.8	0.0	0.0	0	0.0	0.001^s
Obese II (≥30)	0	0.0	0	0.0	0	0.0	0	0.0	

s= significant, ns= not significant, p value reached from ANOVA test

Table VI shows that overweight & obesity was observed among the phenotype A & phenotype B.

IV. Discussion

Polycystic ovary syndrome(PCOS) affects 5-10% of reproductive age women, therefore it is a common endocrine disorder[14]. However, previous report from a variety of different countries demonstrate the diversity in the incidence of PCOS. In this cross sectional study the frequency of PCOS is 37% (27), according to the Rotterdam PCOS criteria. This result indicate that PCOS has reached widespread among the students. Study participants are the girls from one educational instititution who had not sought medical help for any of these symptoms pointing to PCOS. Community based screening among Asian population found prevalence of PCO among 6.3% of reproductive age women aged 15-45 years in Srilanka[10]& 5.6% in China[15], using Rotterdam criteria unlike the higher prevalence observed in our study among younger aged 22-24 years old probably due to smaller group of girls who got enrolled due to more symptoms. Similar study among adolescent & young girls in Mumbai found prevalence of PCO by Rotterdam[16]. The age groups studied in Chinese (20-45 years), South Australian (27-34 years), &Srilankan (15-39 years) population had a wide range whereas our study restricted to medical students within a narrow range(20-25 years) in a same institution which reflect the current situation of PCOS status among the students of Bangladesh is the strength of our study. The prevalence is relatively higher than that reported by most studies mainly due to use of different diagnostic criteria, study settings, age group.

Although many studies have investigated the prevalence of PCOS, there are discrepancies in their results, in part due to the use of various definitions of the syndrome and its subphenotypes, differences between study cohorts, ethnicities, and types of recruitment and sampling.

In our study we found that comparison of our results with those of the other author s is difficult since there are limited data in the literature about PCOS in this age group. Very few studies have done on the normal population to find out the prevalence in the south Asian region. Other study was done only those who had oligomenorrhoea were invited for ultrasound of the ovaries and blood test & remaining women were considered not to have PCOS[6]. Another Indian study the inclusion study was based on menstrual irregularities &/or presence of hirsutism with an mFG score greater than 6 were invited for pelvic ultrasound[17].

In comparison with other studies that have used Rotterdam criteria , among 27 confirmed PCOS in our study , the prevalence of oligo/amenorrhoea observed (88.9%) was similar to that found in Indian (97.62%)[17]& the Srilankan population(95.1%)[6]but much higher compared to that found in south Australian population(23.8%)[6]. This points to a higher prevalence of oligo/amenorrhoea in the Asian scenario.). In the present study, menstrual abnormalities mostly oligomenorrhoea and amenorrhoea are comparable with the finding of who also showed menstrual cycle disturbances among 98% of PCOS women[18]. Hirsutism is considered the best clinical marker of hyperandrogenism; however, the severity of hirsutism varies with ethnicity. In the present study,

hirsutism was observed in 88% of girls among the PCOS group. This finding is in accordance with the finding of Aziz et al. 2006[3]which also showed prevalence of hirsutism among PCOS women between 60 - 80%.

The breakdown of PCOS of medical students in our study revealed most prevalent phenotype is phenotype B (59.3%). In similar other studies the phenotype A,B,C,D of Turkish population 44.09, 14.17, 18.9, and 14.1%[19]; Bulgarian 53.6, 11, 12.8, and 22.6%[20]; United States 58, 13, 14, and 14%[21]; and Iranian 32.1, 46.8, 14.8, and 6.3%[22], respectively. PCOS complete (P + O + H) was the largest group in most reports. Normoandrogenic phenotype (P + O) comprised a significant proportion of PCOS women. Ovulatory phenotype P + H, was the least common group, possibly because these woman are mostly asymptomatic and unlikely to present in Endocrinology outpatient department. Phenotype B is not common to other study. Retrospectively, if all the students of same class would have participated in the study, the prevalence could further reduce the number.

Obesity is a common clinical feature observed in approximately 50% of PCOS women[23]. In our study result shows a high frequency of overweight and obesity in our PCOS population (59.86%). This is consistent with study done by Haider et al. 2012 which showed a frequency of 63.55% and 69% of obesity. This higher incidence of obesity may be attributed to gradual life style change in our population.

It is well documented that PCOS women have a high prevalence of abdominal body fat distribution, even if they are normal-weight, making them more vulnerable to obesity related health problems like diabetes, hypertension and cardiovascular disorders[24].Overweight and obesity were mostly found in phenotype A and phenotype B which is also showed by the other studies[25]. In a similar study, obesity was found to be 28.2 % in Mumbai population[26]. BMI was also found to be higher in phenotype B in a study conducted by Welt et al.2006.

The prevalence of PCOS among obese reproductive-age women has not been well studied. In a recent study from Spain, PCOS was 5-fold more common among unselected premenopausal overweight or obese women seeking advice for weight loss compared to that of the general population (28.3% vs. 5.5%, respectively). In this study, the increased prevalence of PCOS in overweight and obese women was irrespective of the degree of obesity and was independent of the presence or absence of the metabolic syndrome or its features. The study demonstrates the prevalence of PCOS may be markedly increased in overweight and obese women. Same findings was also observed among our study population. Those who had PCOS their(20.54%) BMI was higher than non PCOS group, among the phenotype B it is highest in number (50%). So it can be possible as we collected data from the population of affluent society of Bangladesh, stressful life, inactivity that lead rise to obesity which is responsible for developing PCOS.

In our current study, we found that Family history of PCOS & DM is also more in those who had PCOS. Other studies also demonstrate that PCOS & DM is significantly more prevalent among family members than in the general population. History of diabetes mellitus in first degree relatives of PCOS subjects was 48.75% which was found about 28.2% in a study of Iranian population. One study showed that positive family history of PCOS is present in 21.4% of the patient with PCOS and another study showed 35% of mother and 40% of sister of patient withPCOS are affected by PCOS[3] . The prevalence may be much more higher as significant number of cases has not properly diagnosed. In our study we found 9 among family members of 73 students had family history of PCOS, among them 7 students (25.9%) had PCOS. Around two-third (77.8%) had positive family history of DM, that is much higher than other study.

Our study revealed that hypothyroidism was observed in 7 (25.9%) girls among the PCOS group which was statistically significant. Hypothyroidism also contribute to obesity that is also responsible for development of PCOS[27,28]. There is enough literature support to argue that prevalence of subclinical hypothyroidism/thyroid autoimmunity is increased in women with PCOS patients[29] . Though association between hypothyroidism and PCOS is not yet established. Long-term studies are required to assess the significance of thyroid dysfunction in patients with PCOS

V. Conclusion

Prevalence of PCOS is 37%, predominant Phenotype is B which demonstrates that PCOS is an emerging disorder. This draws attention to the issue of early diagnosis which could provide opportunity to target the group to prevent future morbidities.

Limitation:
This study was conducted in a single hospital where randomization was not done with minimal sample.

Conflict of interest:
There is no conflict of interest.

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