Demographic, clinical and biochemical profile of patients with polycystic ovary syndrome: a study in a tertiary care hospital of Bangladesh

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ABSTRACT

Background: Polycystic ovary syndrome (PCOS) is a common and heterogeneous endocrine disorder, marked by irregular or absent menstrual cycles, excess androgens and polycystic ovaries. The prevalence and diagnosis of PCOS vary depending on the clinical criteria employed, with a prevalence as high as 8-13% when using the Rotterdam criteria. This study was designed to assess the demographic, clinical and biochemical profile of patients with PCOS.

Methods: This cross-sectional study was conducted in the Department of Obstetrics & Gynecology, Dhaka Medical College Hospital, Dhaka, Bangladesh from January 2015 to December 2016. The study included 51 women aged 20-35 years with PCOS. Participants were selected using convenient sampling. Data was collected through a semi-structured questionnaire and analyzed using MS Office tools and SPSS Version 23.0.

Results: The mean age and BMI of our participants were 25.0 ± 32 years and 27.0 ± 3.1 kg/m² respectively. The most common symptom was oligomenorrhoea (74.5%) and the ultrasonographic finding was polycystic ovary (92%). The mean s. testosterone and D2 serum luteinizing hormone (LH) were 1.8 ± 0.9 ng/ml and 12.7 ± 6.7 mIU/mL respectively and s. LH/FSH ratio was increased in 30 (58.8%) patients. The mean fasting insulin, fasting blood glucose and HOMA IR were 27.3 ± 10.7 iU/ml, 5.1 ± 0.8 mmol/L and 4.1 ± 1.3 respectively. Regarding lipid profile status of study subject, 68.60 % had high LDL and 60.60% had high triglyceride level.

Conclusion: The most common clinical feature was oligomenorrhoea and polycystic ovary in ultrasonography. LDL cholesterol and triglycerides were higher in comparison to total cholesterol in PCOS cases.

Key words: Demographic, Clinical Profile Polycystic Ovary Syndrome, Oligomenorrhoea.

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder affecting approximately 7-10% of women of reproductive age worldwide. This condition is characterized by a wide range of androgen-related issues and can result in various reproductive and metabolic abnormalities.¹ The severity of PCOS symptoms is influenced by a combination of genetic and environmental factors.² PCOS is primarily recognized as the leading cause of anovulatory infertility as well as conditions such as hyperandrogenism and hirsutism. PCOS's history dates back to the 18th century when it was initially described by Valisnere as a condition affecting infertile, moderately obese and young married women with distinct ovarian characteristics.³ In 1935, it was further documented as Stein-Leventhal syndrome highlighting the clinical features of amenorrhea, hirsutism, obesity and polycystic-appearing ovaries.⁴ Since then PCOS has garnered increasing attention due to its genetic diversity and the wide array of clinical presentations it encompasses. The diagnosis of PCOS remains a matter of debate due to its heterogeneous in nature which can evolve over a woman's lifetime. Presently the most widely accepted diagnostic criteria for PCOS are the "Rotterdam criteria" established in May 2003. These criteria require the presence of any two of the following three features: 1) oligo/anovulation (O) 2) clinical and/or biochemical hyperandrogenemia (H) and 3) polycystic ovaries identified through ultrasound (P) while also ruling out other known hyperandrogenemia disorders. These criteria give rise to four distinct phenotypes: 1. Phenotype-A (P+H+O) 2. Phenotype-B (P+O) 3. Phenotype-C (H+O) and 4.Phenotype-D (P+H). This classification system aids in characterizing the diverse clinical presentations of PCOS.⁵ Clinical symptoms associated with PCOS encompass menstrual irregularities, hirsutism and frequent infertility or subfertility. Menstrual irregularities in PCOS patients often manifest as prolonged, erratic menstrual bleeding, amenorrhea and oligomenorrhea.⁴ It is important to note that some females with PCOS may exhibit normal menstrual cycles, either with or without anovulation.⁵ Notably, the majority of females presenting with oligomenorrhea and approximately half of those with amenorrhea are diagnosed with PCOS.⁶ Furthermore, most females displaying clinical signs of androgen excess, such as hirsutism, acne or androgenic pattern hair loss, will ultimately receive a PCOS diagnosis.7 Hirsutism, characterized by the excessive growth of terminal hair in a male-pattern distribution, is a common clinical feature of hyperandrogenism observed in a significant proportion of females with PCOS.8 PCOS is associated with various comorbidities. Weight gain leading to overweight and obesity often precedes the clinical manifestations of PCOS. Implementing a healthy lifestyle, including dietary adjustments and exercise therapy, has been demonstrated to reduce weight, enhance insulin sensitivity, decrease abdominal fat, lower testosterone levels and ameliorate hyperandrogenism in females with

PCOS.^{9, 10} Additionally, PCOS is linked to various forms of diabetes, including Type 1, Type 2 and gestational diabetes mellitus.¹⁰ In various study women with type 1 diabetes mellitus were screened for PCOS using the Rotterdam criteria and compared with controls matched for body mass index (BMI).¹¹ So in our study we want to assess the demographic, clinical and biochemical profile of patients with PCOS.

METHODS

This cross-sectional study was carried out in the Department of Obstetrics & Gynecology at Dhaka Medical College Hospital in Dhaka, Bangladesh, spanning from January 2015 to December 2016. The study included a total of 51 women diagnosed with PCOS, aged between 20 to 35 years, who participated as the study patients. Convenient sampling was employed for participant selection and the study received approval from the hospital's ethical committee. Additionally, all participants provided written consent before the commencement of data collection. Demographic and clinical data for all participants were collected and processed using MS Office and analyzed with SPSS version 23.0 as needed for the study.

RESULTS

The age distribution of study participants revealed that the majority of women (56.9%) fell within the 20-25 years age group, while 35.3% and 7.8% were in the 26-30 and 31-35 years age groups, respectively. Regarding the BMI status of the study patients, it was observed that nearly three-fourths of the women (71%) had a BMI of $\geq 25 \text{ kg/}$ m², while the remaining cases (29%) had a BMI falling within the range of 18.5-24.9 kg/m². In terms of waist circumference, hip circumference and the waist-to-hip (W/H) ratio concerning insulin resistance, 23 patients exhibited mean values were in normal range (mean + SD < 0.2) & the remaining 28 participants show high values (>3.2) (Table I). In this study, oligomenorrhea (74.50%) is the most prevalent clinical symptom & the percentage of other features like hirsutism, acanthosis nigricans, amenorrhea, acne and hypertension were shown in Figure-1. Regarding the ultrasonography (USG) findings among the study patients, it was observed that the majority of women (39.21%) had polycystic ovaries, while

27.45% had enlarged ovaries (13.0 + 1.5 cm3), and 33.34% were found to have normal ovarian characteristics. In terms of laboratory values, the mean serum testosterone level was 1.8 (\pm 0.9) ng/ml. The mean D2 serum FSH and D2 serum LH levels were 5.7 ± 2.0 IU/L and $12.7(\pm6.7)$ mIU/mL respectively (Table II). Regarding the status of insulin resistance about 54.9% patients show high HOMA IR (>3.2) & 43.2% show impaired fasting glucose (Table III). An analysis of the serum LH/FSH ratio in the study patients revealed an increase ratio among 30 patients (58.8% of total cases). On lipid profile analysis 54.9% of the patients had HDL levels below 40 mg/dl while 45.09% had HDL levels above 40 mg/dl. Additionally, the majority of patients had total cholesterol levels less than 200 mg/dl accounting for 83.40% of the cases, while 17.60% had total cholesterol levels exceeding 200 mg/dl. Similarly for triglycerides, most patients (83.40%) had levels below 200 mg/dl (Table V).

Table I. Waist, hip circumference and W/H ratio
with insulin resistance of patients. (N=51)

Variables	Insulin resistance	
-	<3.2 (normal)	>3.2 (high)
	(n=23)	(n=28)
	Mean+SD	Mean+SD
Waist circumference (cm)	82.4+6.7	86.6+3.9
Hip circumference (cm)	99.4+4.6	101.4+5.6
W/H Ratio	0.80+0.38	0.83+0.18

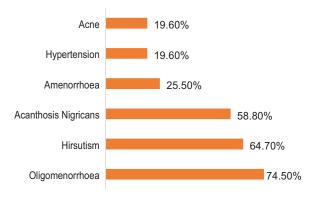




Table II. Hormone profile variation in patients with

 Insulin Resistance. (N=51)

Variables	<3.2	>3.2	Р	
	(n=23)	(n=28)	value	
Mean+SDMean+SD				
Serum testosterone (ng/n	nl) 2.2+1.0	2.8+0.8	0.021s	
D2 Serum FSH (IU/L)	4.8+1.9	5.2+2.1	0.483ns	
D2 Serum LH (mIU/mL)	11.2+3.3	14.5+5.0	0.006s	
Fasting insulin (μ U/ml)	26.8+12.1	32.8+8.3	0.041s	

Table III. Distribution of insulin resistance. (N=51)

Variables	n	%
Fasting insulin ((µU/ml)		
<25	21	41.20%
>25	30	58.80%
Mean+SD	27.3+10.7	
Fasting blood glucose (mmol/L)		
3.9-6.0	29	56.9%
6.1-6.9	22	43.2%
Mean+SD	5.1 + 0.8	
HOMAIR		
<3.2	23	45.1%
>3.2	28	54.9%
Mean+SD	4.1+1.3	

Table IV. Distribution of fasting lipid profile. (N=51)			
Fasting lipid profile	n	%	
Total cholesterol (mg/dl)			
<200	42	83.40%	
>200	9	17.60%	
Triglycerides (mg/dl)			
<150	20	39.20%	
>150	31	60.80%	
HDL (mg/dl)			
<40	28	54.90%	
>40	23	45.09%	
LDL (mg/dl)			
<100	16	31.40%	
>100	35	68.60%	

HDL: High density lipoprotein, LDL: Low density lipoprotein

DISCUSSION

In this study, among the age distribution most women (56.9%) were in the 20-25 year's age group, while 35.3% and 7.8% were in the 26-30 years and 31-35 year's age groups respectively. About 71% of the women had a BMI of e" 25 kg/m^2 , while the remaining 29% fell within the 18.5-24.9 kg/m² BMI range. Yildir et al study found a mean BMI of 26.12±5.68 kg/m².13 Among the study subjects 23 cases revealed normal finding of waist circumference, hip circumference & waist-hip ratio & the remaining show high values. Cakir E et al study reported W/H Ratio of 0.84±0.5 which was very close to our study.¹⁴ In this study oligomenorrhea was acommon clinical profile affecting 74.50% of patients. Hirsutism (64.70%), acanthosis nigricans (58.80%), amenorrhea (25.50%), acne (19.60%) and hypertension (19.60%) were also observed. In another study by Azziz R et al were reported that 70.0% of patients with PCOS presented with hirsutism and 15.25% had acne, findings are consistent with the results of our study.¹⁵ Regarding the USG findings we found the highest number of women (92.20%) had polycystic ovaries, 27.50% had enlarged ovaries (13.0+1.5 cm³) and 33.30% had normal ovaries. In terms of laboratory values, the mean serum testosterone level was 1.8 (\pm 0.9) ng/ml. The mean D2 serum FSH and D2 serum LH levels were 5.7±2.0 IU/L and $12.7(\pm 6.7)$ mIU/mL respectively. However, the difference in D2 serum FSH was not statistically significant (p > 0.05). Yousouf et al reported elevated serum testosterone in 57.7% of PCOS patients, which differs from the findings of our study.¹⁶ Sun et al found higher levels of serum testosterone with mean values of 48.48±19.53 ng/dl.¹⁷ Similarly Cakir et al also found higher levels of serum testosterone which aligns with the results of our study.¹⁴ In this study, PCOS patients with HOMA-IR >3.2 exhibited higher levels of serum testosterone, D2 serum LH, and fasting serum insulin than the PCOS patients with HOMA-IR <3.2. In our study the serum LH/FSH ratio was increased in 30 cases, accounting for 58.8% of study subject which are consistent with the findings of Sun et al, who also reported significantly higher levels of LH and LH/FSH ratio.¹⁷ Moreover, Begum F et al found that 51.0% of PCOS patients had LH/FSH ratio greater than 2.18 and Razzak A et al reported that 64.0% of PCOS patients had LH/FSH ratio greater than 2.19 Regarding distribution of insulin resistance, the mean fasting

insulin level was found 27.3 \pm 10.7iU/ml, fasting blood glucose was 5.1 \pm 0.8 mmol/L & the mean calculated HOMA IR was 4.1 \pm 1.3. Similar finding was observed in *Begum F et al* study, where showed mean fasting blood glucose level 5.98 \pm 1.08 mmol/L with a p-value of less than 0.01.¹⁸ Majority (54.9%) of study subjects shows HDL levels below 40, total cholesterol levels less than 200 accounting for 83.40% and for triglycerides most patients (83.40%) had levels below 200. But most (68.60%) of the study participant showed high LDL level. *Enzevaie et al* demonstrated that the triglyceride level was 159 in most of the cases.²⁰ However, total cholesterol levels and HDL levels did not show significant differences in their study which aligns with the findings of our study.

Conclusion

Among patients with PCOS oligomenorrhea and the presence of polycystic ovaries are the prevailing clinical feature and ultrasonographic findings respectively. Furthermore it is noteworthy that PCOS patients typically exhibit higher levels of LDL cholesterol and triglycerides. These findings shed light on the clinical characteristics and potential metabolic implications of PCOS offering valuable insights into its diagnosis and management.

Authors' contribution: BKA, NAH, RKA planned the research and collected the data. BKA, SIR, SCB analyzed data and drafted manuscript. BKA, RC supervised the whole process. All authors read and approved the final version for publication.

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Conflicts of interest: Nothing to declare.

REFERENCES

- Cloyton RN, Ogelen V, Hodgkinson J, Worswick L, Roden D, Dyer S, et al. How common are PCO in normal women and what is their significance for the fertility of the population. Clin Endocrinol 1992; 37:127-34.
- Knochenhauer ES, Key TJ, Kahsar-Miler M, Waygoner W, BOOTS LR, Azziz KI. Prevalance of PCOS in black and white women of the southeastern United States: a prospective study. J Clin Endocrinal metab 1998; 83:3078-82.
- Insler V, Lunenfeld B. PCOD: A challenge and controversy. Gynecol Endocrinol 1990; 4:51-70.
- Stein IF, Leventhal ML. Amenorrhoea associated with bilateral PCO. AM J Obstet Gynecol 1935; 29:181-91.

- Rotterdam ESHRE/ASRM sponsored PCOS consensus workshop group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to PCOS. Hum Reprod 2004; 19:41.
- Farquhar C. Introduction and history of polycystic ovary syndrome. In: Kovacs G, Norman R, editors. Polycystic Ovary Syndrome. 2nd ed. Cambridge, UK: Cambridge University Press; 2007. p 4-24.
- Balen AH, Conway GS, Kaltsas G, Techatrasak K, Manning PJ, West C, et al. Polycystic ovary syndrome: the spectrum of the disorder in 1741 patients. Hum Reprod 1995; 10 (8):2107-11.
- Hart R, Hickey M, Franks S. Definitions, prevalence and symptoms of poly-cystic ovaries and polycystic ovary syndrome. Best Pract Res Clin Obstet Gynaecol 2004; 18 (5):671-83.
- Azziz R, Sanchez LA, Knochenhauer ES, Moran C, Lazenby J, Stephens KC, et al. Androgen excess in women: experience with over 1000 consecutive patients. J Clin Endocrinol Metab 2004; 89(2):453-62.
- Fauser BCJM, Tarlatzis BC, Rebar RW, Legro RS, Balen AH, Lobo R, et al. Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS consensus workshop group. Fertil Steril 2012; 97(1):28-38.
- Salehi M, Bravo-Vera R, Sheikh A, Gouller A, Poretsky L. Pathogenesis of polycystic ovary syndrome: what is the role of obesity? Metab Clin Exp 2004; 53:358-76.
- Amisi C, Mputu L, Mboloko E, Bieleli E, Pozzili P. Biological insulin resistance in Congolese woman with polycystic ovary syndrome. Gynecol Obstet Fertil 2013;41(12):707-10.

- Yildir IC, Kutluturk F, Tasliyurt T, Yelken BM, Acu B, Beyhan M. Insulin resistance and cardiovascular risk factors in women with PCOS who have normal glucose tolerance test. Gynecological Endocrinology 2013;29 (2):148-51.
- Cakir E, Topaloglu O, Bozkurt CN, Bayraktar KB, Gungune^o A, Arslan SM, et al. Insulin-like growth factor 1, liver enzymes, and insulin resistance in patients with PCOS and hirsutism. Turk J Med Sci 2014;44(5):781-6.
- Azziz R, Carmina E, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Futterweit W, et al. The Androgen Excess and PCOS Society criteria for the polycystic ovary syndrome: the complete task force report. Fertil Steri 2009; 91:456-88.
- Yousouf R, Khan M, Kounsar Z, Ahangar S, Lone WA. Polycystic ovarian syndrome: Clinical Correlation with Biochemical Status. Surgical Science 2012; 3(5):245-8.
- Sun X, Wu, Zhou Y, Yu X, Zhang W. Evaluation of Apelin and Insulin Resistance in Patients with PCOS and Therapeutic Effect of Drospirenone- Ethinylestradiol Plus Metformin. Med Sci Monit 2015; 21: 2547-52.
- Begum F. Clinical and Hormonal Profile of Polycystic Ovary Syndrome. South Asian Federation of Obstetrics and Gynecology 2009; 1(2):22-5.
- Razzak A, Nadak A, Tace A. Polycystic ovarian syndrome: The Correlation between the LH: FSH and Disease Manifestation. Middle East Fertility Society Journal 2007; 12(1):35-40.
- Enzevaie A, Salehpour S, Tohidi M, Saharkhiz N. Suclinical hypothyroidism and insulin resistance in polycystic ovary syndrome: is there a relationship. Iran J Reprod Med 2014; 12(7):481-6.