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# Association of metabolic syndrome with different phenotypes of polycystic ovarian syndrome among Filipino women in a tertiary hospital: A retrospective cohort study

Maria Anjelette Patricia F. Belen<sup>1</sup>, Susana S. Lao<sup>1</sup>

## Abstract:

**INTRODUCTION:** Polycystic ovarian syndrome (PCOS) is a common endocrinopathy affecting women during reproductive age. Women affected by PCOS generally have a higher risk of developing Metabolic syndrome (MetS). MetS on each phenotype of PCOS reflects some phenotypes with worse metabolic profiles and a higher risk of developing long-term complications in women with PCOS.

**OBJECTIVE:** To determine the association of MetS with different phenotypes of PCOS among Filipino women in a tertiary hospital.

**MATERIALS AND METHODOLOGY:** This is a retrospective cohort study of 154 women in a tertiary hospital, both private and service divisions.

**RESULTS:** A total of 154 patients with PCOS were analyzed in this study: 67 (43.51%) Phenotype A, 25 (16.23%) Phenotype B, 3 (1.95%) Phenotype C, and 59 (38.31%) phenotype D. The prevalence of MetS in PCOS was 69.48%, with no significant difference statistically between phenotypes. MetS was most prevalent in Phenotype A (74.63%) and least prevalent in phenotype D (62.71%). Among Filipino women with PCOS, Phenotype A had a 2.5 times increased risk of developing MetS compared to Phenotype D.

**CONCLUSION:** Phenotype A is the most common phenotype and has the highest prevalence in developing metabolic changes. Increasing body mass index and age played significant roles in elevating the risk of developing MetS. Early detection of MetS in all phenotypes of PCOS can aid in preventing the development of long-term complications such as cardiovascular disease and diabetes mellitus type II.

## Keywords:

Metabolic syndrome, polycystic ovarian syndrome phenotypes, polycystic ovarian syndrome

<sup>1</sup>Department of Obstetrics and Gynecology, Chinese General Hospital and Medical Center, Manila, Philippines

**Address for correspondence:**  
Dr. Maria Anjelette Patricia F. Belen,  
No. 1773 Uranus Cor Jupiter St., ADB Subdivision, Del Remedio, San Pablo City, Laguna, Philippines.  
E-mail: [anjelette.belen@gmail.com](mailto:anjelette.belen@gmail.com)

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## Introduction

Polycystic ovarian syndrome (PCOS) is a common endocrinopathy affecting women during reproductive age.<sup>[1]</sup> PCOS is a complex multigenic disorder that is affected by various influences such as diet and lifestyle factors.

The 2003 ESHRE/ASRM Rotterdam criteria are one of the three consensuses that refine the

diagnostic criteria of PCOS, and it is the most commonly used criteria in clinical practice. The Rotterdam criteria established the diagnosis of PCOS, which include the presence of two out of three features: (1) Oligo- or anovulation or menstrual dysfunction, (2) Clinical and/or biochemical signs of hyperandrogenism, or (3) polycystic ovaries on ultrasound (presence of 25 or more follicles in one or both of the ovaries or ovarian volume  $\geq 10$  cm<sup>3</sup>).<sup>[2]</sup>

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PCOS is a complex endocrine disorder that presents different phenotypes. The four phenotypes of PCOS include: A: Oligo-anovulation or anovulation + clinical and/or biochemical hyperandrogenism + polycystic ovaries; B: Oligo-anovulation or anovulation + clinical and/or biochemical hyperandrogenism; C: Clinical and/or biochemical hyperandrogenism + polycystic ovaries; D: Oligo-anovulation or anovulation + polycystic ovaries.<sup>[3]</sup>

PCOS has been identified as one of the causes of significant morbidity in women.<sup>[4]</sup> The risk of developing reproductive abnormalities such as infertility, abnormal uterine bleeding, and cardio-metabolic disorders such as obesity, insulin resistance, diabetes mellitus, and cardiovascular disease is increased in women with PCOS. Women affected by PCOS generally have a higher risk of developing metabolic disorders. The metabolic disorders of PCOS are related to hyperandrogenism and hyperinsulinemia.<sup>[3]</sup> Metabolic Syndrome (MetS) is composed of interrelated risk factors of metabolic origin that promote the risk of serious diseases such as cardiovascular disease and diabetes mellitus.<sup>[5]</sup> The diagnosis of MetS is considered when at least three of the following criteria are present: waist circumference ( $\geq 80$  cm in Asian women), hypertension (systolic blood pressure (BP)  $\geq 130$  mmHg and/or diastolic  $\geq 85$  mmHg), high levels of blood glucose (fasting level  $\geq 110$ – $126$  mg/dL) and 2 h (140–199 mg/dl) from oral glucose tolerance test (OGTT), high triglyceride levels ( $\geq 150$  mg/dL) and a reduction in high-density lipoprotein (HDL) cholesterol ( $<50$  mg/dL).<sup>[3]</sup>

Women with PCOS have the prevalence of MetS that is almost twice as great as the general population of women.<sup>[6,7]</sup> This increase the risk of cardiovascular disease by seven times.<sup>[8]</sup> The study on MetS on each phenotype of PCOS reveals some phenotypes with worse metabolic profiles and with an increasing number of harmful results to patients' health.<sup>[1,9]</sup> Due to the high incidence of MetS among PCOS patients, this study aims to determine the association of MetS with each phenotype of PCOS in the local setting. This study may help in counseling patients with PCOS by determining the possible risk of having MetS based solely on their PCOS phenotype.

## Objectives of the study

### General objective

To determine the association of MetS with different phenotypes of PCOS among Filipino women in a tertiary hospital.

### Specific objective

1. To determine the incidence of MetS among PCOS patients in a tertiary hospital.

2. To identify the prevalence of each PCOS phenotype and the most common phenotype of PCOS in a tertiary hospital
3. To determine the most common phenotype of PCOS presenting with MetS
4. To determine the association of BMI of PCOS patients with MetS
5. To determine which among the different components of MetS is present in each PCOS phenotype in a tertiary hospital.

## Materials and Methodology

### Study design

This is a retrospective cohort study.

### Setting of the study

The study was conducted at a tertiary hospital.

### Study subject and target population

#### Inclusion criteria

1. Women with PCOS aged 18–39 years old in a tertiary hospital
2. Patients who are diagnosed with PCOS according to the 2003 Rotterdam criteria.

#### Exclusion criteria

1. Patients who were on anti-hypertensive, lipid-lowering drugs, and hypoglycemic medications
2. Pregnant women
3. Medical comorbidities include hypertension, heart disease, thyroid disease, kidney disease, diabetes mellitus, hyperprolactinemia, and other causes of hyperandrogenism, such as congenital adrenal hyperplasia, Cushing's syndrome, adrenal neoplasia, or virilizing ovarian tumors.

### Sample size

A minimum of 154 female patients in a tertiary hospital diagnosed with PCOS aged 18–39 years old were included in the study. Using Epi info 7, 22.3% of the sample size was computed using a 95% confidence level, 80% power, 6.5 with MetS among those without hirsutism, and 22.3% among those with hirsutism based on the study of Neves *et al.*<sup>[1]</sup> 2014. Sample size formula and computation [Figure 1].

### Data collection procedure

The data collection was conducted in a tertiary hospital from March 20, 2021 to December 30, 2021. Data were gathered through chart review. Data were collected using a collecting tool that included the following: patient's age, height, weight, body mass index (BMI), BP, waist circumference, history of menstrual irregularity, presence of hyperandrogenism, transvaginal/transrectal

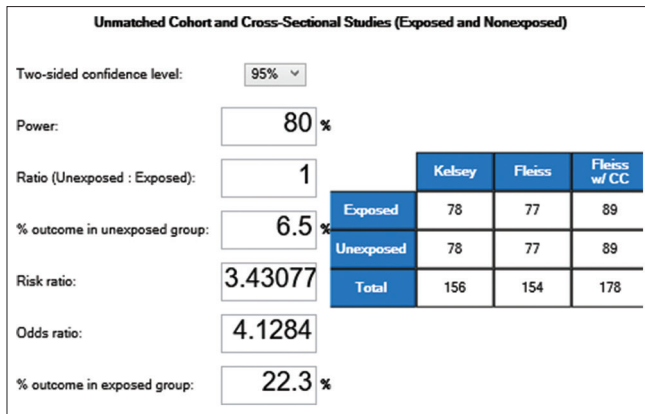


Figure 1: Sample size formula and computation

ultrasound, lipid profile (triglyceride and HDL), and 75 g OGTT results [Figure 2]. Patients included in the study were those who were diagnosed with PCOS from January 2018 to February 2021 in a tertiary hospital, both Private and Service divisions.

### Data management and statistical tools

Descriptive statistics were used to summarize the general and clinical characteristics of the patients. Categorical variables were presented as frequency and proportion. Shapiro–Wilk was used to test the normality assumption of continuous variables. Continuous quantitative data that met the normality assumption were summarized using mean and standard deviation, while those that did not were described using median and interquartile range (IQR). Continuous variables that satisfied the dual assumptions of normal distribution and variance homogeneity were compared using one-way Analysis of variance. If both assumptions were violated, the nonparametric Kruskal–Wallis *h*-test was used for comparison. Fisher’s exact test was used to compare categorical variables with <5% cell expected percentages.

Logistic regression was used to determine the factors associated with MetS. Odds ratio and its corresponding 95% confidence interval (CI) was reported.

The null hypothesis was rejected at 0.05 $\alpha$ -level of significance. STATA version 15.0 (StataCorp SE, College Station, Texas, USA) was used for data analysis.

### Results

A total of 154 patients with PCOS were analyzed in this study: Sixty-seven (43.51%) Phenotype A, 25 (16.23%) Phenotype B, 3 (1.95%) Phenotype C, and 59 (38.31%) Phenotype D [Table 1]. The median age was 25 years (22 being the youngest and 28 being the oldest), and the mean BMI was 27.51  $\pm$  3.51 kg/m<sup>2</sup>. Many patients belong to the age group 20–24 years (65% or 42.21%)

Figure 2: Data Collecting Tool

and 25–29 years (65% or 42.21%), as well as with BMI of 25–29.9 kg/m<sup>2</sup> (74% or 48.05%). Most of the patients (94% or 61.04%) had mild Ferriman–Gallwey scores, including all patients with Phenotypes B and C and majority (66% or 98.51%) of those with phenotype A.

The median waist circumference was 104 cm (IQR 88–117), and the median waist circumference was higher in Phenotypes A and B compared to C and D (113 cm and 110 cm versus 92 cm, respectively, *P* < 0.001). Subsequently, the majority (85.71%) of the patients had a waist circumference of >80 cm. Most of the patients had a systolic BP <130 mmHg (129% or 83.77%) and a diastolic BP of <85 mmHg (131% or 85.06%). Only 25 (16.23%) patients were hypertensive.

The median triglyceride levels (151 mg/dL, IQR 127–156) and fasting glucose (104.08, IQR 94.4–120) were significantly evident in patients with phenotypes A (46% or 68.66%) and C (3% or 100%), respectively. For fasting glucose, the median value of Phenotype A was highest at 110, compared to Phenotype B and C at 100, and Phenotype D at 99 (*P* = 0.028). 2-h OGTT was not significantly different across the phenotypes [Table 2].

There were 107 patients considered to have MetS. Overall, the prevalence of MetS was 69.48% (95% CI 61.6% to

**Table 1: Characteristics of patients with polycystic ovarian syndrome (n=154)**

	All (n=154)	A (n=67; 43.51%)	B (n=25; 16.23%)	C (n=3; 1.95%)	D (n=59; 38.31%)	P
	Median (IQR); frequency (%); mean±SD					
Age (years)	25 (22-28)	24 (22-26)	27 (25-28)	26 (25-33)	26 (22-28)	
18-19	3 (1.95)	1 (1.49)	1 (4)	0	1 (1.69)	0.108*
20-24	65 (42.21)	34 (50.75)	5 (20)	0	26 (44.07)	
25-29	65 (42.21)	28 (41.79)	16 (64)	2 (66.67)	19 (32.2)	
30-34	20 (12.99)	4 (5.97)	3 (12)	1 (33.33)	12 (20.34)	
≥35	1 (0.65)	0	0	0	1 (1.69)	
BMI (kg/m <sup>2</sup> )	27.51±3.51	28.09±3.33	27.98±2.91	25.73±1.10	26.74±3.89	
<18.5	0	0	0	0	0	0.114†
18.5-22.9	19 (12.34)	4 (5.97)	1 (4)	0	14 (23.73)	
23-24.9	15 (9.74)	7 (10.45)	4 (16)	0	4 (6.78)	
25-29.9	74 (48.05)	32 (47.76)	12 (48)	3 (100)	27 (45.76)	
≥30	15 (9.74)	24 (35.82)	8 (32)	0	14 (23.73)	
Ferriman-Gallwey score	9 (0-11)	11 (10-13)	9 (9-12)	10 (8-11)	0	
None	59 (38.31)	0	0	0	59 (100)	<0.001*
Mild	94 (61.04)	66 (98.51)	25 (100)	3 (100)	0	
Moderate	1 (0.65)	1 (1.49)	0	0	0	

Statistical tests used: \*Kruskal-Wallis H-test, †One-way ANOVA test, ‡Fisher's exact test. Boldface indicates statistical significance. Phenotype A: Oligo-anovulation + hyperandrogenism + polycystic ovaries, Phenotype B: Oligo-anovulation+hyperandrogenism, Phenotype C: Hyperandrogenism+polycystic ovaries, Phenotype D: Oligo-ovulation or anovulation + polycystic ovaries. SD: Standard deviation, IQR: Interquartile range, BMI: Body mass index

76.6%). This was most pronounced in Phenotype A at 74.63% (95% CI 62.4% to 84.5%) but similarly prevalent in Phenotype B at 72%, Phenotype C at 66.67%, and Phenotype D at 62.71%.

For every unit increase in age, the odds of having MetS increase by 1.34 times (crude odds ratio [cOR] 1.342, 95% CI 1.18–1.52,  $P < 0.001$ ) and for every unit increase in BMI, the odds of MetS doubles [cOR 1.99, 95% CI 1.61–2.48,  $P < 0.001$ , Table 3].

After adjusting for age, BMI, and both age and BMI, and using Phenotype D as reference, there was no association between Phenotypes B and C with MetS. Meanwhile, Phenotype A was 2.5 times as likely (adjusted odds ratio 2.484, 95% CI 1.05–5.87,  $P = 0.038$ ) to develop MetS compared to Phenotype D, after adjusting for age, but not when adjusted with BMI [Table 4].

## Discussion

In this retrospective cohort study, a total of 154 charts were gathered and reviewed, 107 of which fulfilled the criteria of MetS. It was found that Phenotype A (43.51%) and nonhyperandrogenic, Phenotype D (38.31%) were the most frequent phenotypes, followed by phenotype B (16.23%) and by the least common, phenotype C (1.95%). In contrast to the study of Tavares *et al.*, they evaluated 111 Brazilian women with PCOS; phenotypes A and B were the most diagnosed phenotype.<sup>[3]</sup> In the study of Ladrón de Guevara *et al.*, the least prevalent among western women with PCOS is phenotype D. This variability in the prevalence of nonhyperandrogenic phenotype D among the studies, may be due to subjective clinical evaluation of hirsutism

which is the most reliable marker of androgen excess.<sup>[8]</sup> According to the study of Wild *et al.*, an interobserver variability of about 50% was found on the Ferriman Gallwey Score, depending on the area being examined.<sup>[10]</sup> However, the Ferriman Gallwey score is still a standard scale used in clinical practice because it is cost-effective and convenient to use.<sup>[11]</sup> Based on the results of this study, most of the patients in each phenotype have mild scores in the Ferriman Gallwey Scale. Phenotype A (98.51%) being the most frequent hyperandrogenic phenotype. According to the study of Neves *et al.*, the metabolic risk is tripled with the presence of hirsutism when compared to those without hirsutism.<sup>[1]</sup>

The prevalence of MetS in PCOS was 69.48%, with no significant difference statistically between phenotypes. In this study, it was found that MetS was most prevalent in Phenotype A (74.63%) and least prevalent in nonhyperandrogenic phenotype D (62.71%). It showed an increased risk of 2.5 times as likely to develop MetS in Phenotype A in relation to Phenotype D among Filipino women with PCOS in this study.

In the evaluation of parameters of MetS, it was evident in this study that majority of BMI in all phenotypes were mostly overweight and obese (ranging from 25 to 29.9) based on WHO Asian Criteria. This study revealed that there is an increased risk of developing MetS for every unit increased in BMI. Most of the PCOS patients (85.71%) included in the study have a waist circumference >80 cm, and majority of which was apparent in Phenotypes A. This may explain the higher prevalence of metabolic changes encountered within this phenotype. This result indicates the significant role of waist circumference or abdominal obesity, which can be reflected to the

**Table 2: Metabolic syndrome parameters, overall and across polycystic ovarian syndrome phenotypes (n=154)**

	All (n=154)	A (n=67; 43.51%)	B (n=25; 16.23%)	C (n=3; 1.95%)	D (n=59; 38.31%)	P
	Median (IQR); frequency (%); mean±SD					
Waist circumference (cm)	104 (88-117)	113 (98-120)	110 (90-114)	92 (86-95)	92 (78-110)	<0.001*
<80	22 (14.29)	5 (7.46)	1 (4)	0	16 (27.12)	0.007‡
≥80	132 (85.71)	62 (92.54)	24 (96)	3 (100)	43 (72.88)	
SBP (mmHg)						
<130	129 (83.77)	58 (86.57)	24 (96)	2 (66.67)	45 (76.27)	0.067‡
≥130	25 (16.23)	9 (13.43)	1 (4)	1 (33.33)	14 (23.73)	
DBP (mmHg)						
<85	131 (85.06)	60 (89.55)	24 (96)	2 (66.67)	45 (76.27)	0.034‡
≥85	23 (14.94)	7 (10.45)	1 (4)	1 (33.33)	14 (23.73)	
Hypertension	25 (16.23)	9 (13.43)	1 (4)	1 (33.33)	14 (23.73)	0.067‡
Triglycerides (mg/dL)	151 (127-156)	152 (134.09-157)	153 (141-156)	74.34 (65.49-150)	134 (107-153)	0.001*
<150	66 (42.86)	21 (31.34)	8 (32)	2 (66.67)	35 (59.32)	0.005‡
≥150	88 (57.14)	46 (68.66)	17 (68)	1 (33.33)	24 (40.68)	
HDL cholesterol (mg/dL)	38 (34.5-51)	37 (34-49)	37 (32-52)	48.12 (36-63.71)	44.8 (36-57)	0.158*
<50	113 (73.38)	52 (77.61)	18 (72)	2 (66.67)	41 (69.49)	0.674‡
≥50	41 (26.62)	15 (22.39)	7 (28)	1 (33.33)	18 (30.51)	
Fasting glucose	104.08 (94.4-120)	110 (99-123)	100 (98-115)	100 (94.4-101.8)	99 (89.64-115)	0.028*
≤110	89 (57.79)	32 (47.76)	14 (56)	3 (100)	40 (67.8)	0.269‡
≥110-126	47 (30.52)	24 (35.82)	9 (36)	0	14 (23.73)	
>126	18 (11.69)	11 (16.42)	2 (8)	0	5 (8.47)	
2 h OGTT	134 (110.09-158.5)	139 (120-187)	141 (123-147)	123 (95-135.72)	123 (100-156)	0.198*
<140	88 (57.14)	34 (50.75)	12 (48)	3 (100)	39 (66.1)	0.383‡
≥140-199	52 (33.77)	27 (40.3)	10 (40)	0	15 (25.42)	
>199	14 (9.09)	6 (8.96)	3 (12)	0	5 (8.47)	
Metabolic syndrome, frequency; prevalence (95% CI)	107; 69.48 (61.6-76.6)	50; 74.63 (62.4-84.5)	18; 72 (50.6-87.9)	2; 66.67 (9.4-99.2)	37; 62.71 (49.1-75.0)	0.504‡

Statistical tests used: \*Kruskal-Wallis H-test, †Fisher's exact test. Boldface indicates statistical significance. Phenotype A: Oligo-anovulation + hyperandrogenism + polycystic ovaries, Phenotype B: Oligo-anovulation + hyperandrogenism, Phenotype C: Hyperandrogenism + polycystic ovaries, Phenotype D: Oligo-ovulation or anovulation + polycystic ovaries. SD: Standard deviation, IQR: Interquartile range, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, HDL: High-density lipoprotein, OGTT: Oral glucose tolerance test, CI: Confidence interval

**Table 3: Factors associated with metabolic syndrome (n=154)**

	COR (95% CI)	P
Age (years)	1.342 (1.18-1.52)	<0.001
BMI (kg/m <sup>2</sup> )	1.999 (1.61-2.48)	<0.001
Mild-to-moderate Ferriman-Gallwey Score (reference: None)	1.665 (0.83-3.35)	0.152
Menstrual irregularity and amenorrhea	1.141 (0.10-12.90)	0.915
Hyperandrogenism	1.665 (0.83-3.34)	0.152
Polycystic ovaries	0.865 (0.33-2.24)	0.765
Phenotype		
A	1.749 (0.82-3.75)	0.151
B	1.529 (0.55-4.24)	0.415
C	1.189 (0.10-13.89)	0.890
D	Reference (1.00)	-

Boldface indicates statistical significance. COR: Crude odds ratio, BMI: Body mass index, CI: Confidence interval

alterations in metabolic parameters. Elevated triglyceride levels >150 mg/dL (68.66%) and increased fasting glucose were more prevalent in phenotype A. Based on the study of Sachdeva *et al.*, phenotype A was found to have a higher prevalence of obesity, hyperandrogenism,

insulin resistance, and deranged lipid profile, which may eventually lead to long-term health complications such as increased cardiovascular risk, higher chances of having Type II diabetes mellitus.<sup>[12]</sup>

The increase in risk factors for cardiovascular diseases with PCOS warrants screening for additional risk factors, including sedentary lifestyle, dyslipidemia, hypertension, and impaired glucose tolerance.<sup>[13]</sup> Type II diabetes mellitus and impaired glucose tolerance are increased in patients with PCOS, which is also exacerbated by obesity.<sup>[14,15]</sup> The International evidence-based guideline for the assessment and management of PCOS, published in 2018, recommends that all women with PCOS be evaluated at least at the initial consultation and on a minimum of 2-year interval thereafter with OGTT, Glycated hemoglobin, or fasting glucose. OGTT is recommended for those high-risk women with BMI >25 kg/m<sup>2</sup> in the Asian population.<sup>[6]</sup>

In the observational study of Neves *et al.*, results revealed that differences in ethnicity, diet, and environment can also lead to alterations in metabolic parameters.<sup>[1]</sup> In

**Table 4: Association of phenotypes with metabolic syndrome**

	OR (adjusted for age)	P	OR (adjusted for BMI)	P	OR (adjusted for age and BMI*)	P
Phenotype (reference: phenotype D)						
A	2.484 (1.05-5.87)	0.038	0.748 (0.25-2.27)	0.608	0.994 (0.31-3.18)	0.992
B	1.262 (0.40-3.97)	0.690	0.579 (0.13-2.50)	0.464	0.588 (0.13-2.63)	0.487
C	0.563 (0.04-8.35)	0.676	1.397 (0.10-18.92)	0.801	0.956 (0.07-13.46)	0.973

\*Age and BMI are directly correlated in this sample. OR: Odds ratio, BMI: Body mass index

the same study, it revealed that the main determinants of the high prevalence of MetS in PCOS patients were the incidence of obesity and the impact of age. This was evident in this study that as BMI increases, it doubles the risk of developing MetS, and as age advances, risk increases by 1.34 times. In contrast to the study done by Mata and Jasul., which revealed that the prevalence of MetS was higher in patients with normal BMI with hypertension as the most prevalent parameter of MetS.<sup>[16]</sup> The study by Legro *et al.* stated that obesity and age substantially increase metabolic risk, and PCOS is responsible for the higher incidence of diabetes mellitus and insulin resistance among patients with normal BMI.<sup>[13]</sup> Therefore, screening and diagnosis for MetS should not only be limited to patients with higher BMI.

### Limitations of the study and recommendation

A limitation of the study is the data gathering which focused only on the clinical evaluation and metabolic parameters of women with PCOS. Further prospective studies are recommended to determine which clinical, metabolic, and hormonal components of women with PCOS may be considered predictive factors for the diagnosis of MetS. Future researches may focus in associating parity as well as the development of MetS after pregnancy since there is an additional body mass gain that may lead to worsening of metabolic parameters in PCOS women.

This study only included a small scale of subjects and was limited to those individuals who have complete anthropometric measurements and metabolic parameters workup. It is recommended to have further studies which will include a larger sample size that can be reflective of the general Filipino women with PCOS.

### Conclusion

A prevalence of 69.48% was found in the association between PCOS phenotypes and MetS. Phenotype A is the most common phenotype and has the highest prevalence of developing metabolic changes. Increasing BMI and age played significant roles in elevating the risk of developing MetS. Early detection of MetS in all phenotypes of PCOS can aid in preventing the development of long-term complications such as cardiovascular disease, and diabetes mellitus type II. Large-scale investigations in evaluating long-term outcomes are needed to determine the impact of MetS in the background of PCOS.

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Nil.

### Conflicts of interest

There are no conflicts of interest.

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