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Evaluation of sonographic endometrial findings among patients with polycystic ovarian syndrome: A retrospective study in a local tertiary hospital

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Abstract:

BACKGROUND AND OBJECTIVE: Polycystic ovarian syndrome (PCOS) is a complex disease associated with endometrial lesions. Local data on endometrial findings associated with PCOS are limited. This study aimed to determine the local prevalence and spectrum of endometrial findings and their association with clinical factors among Filipino women with PCOS.

METHODOLOGY: This is a retrospective review of women aged 18–40 years old seen at a local tertiary hospital from January 2016 to December 2020 with ultrasound findings of polycystic ovaries based on Rotterdam criteria. The clinical data and ultrasound findings/impressions were reviewed. Histopathologic results when available were retrieved. Data were analyzed using descriptive statistics; abnormal endometrial findings were associated with clinical factors using binary logistic regression analysis.

RESULTS: A total of 177 women were included in the study, and 39 (22%) had abnormal endometrial findings by ultrasound including thickened endometrium (14.7%), polyp (5.1%), submucous myoma (1.1%), and malignancy (1.1%). Ultrasound findings that were significantly common with abnormal endometrium included thickening, nonuniform echogenicity, and the presence of vascularity. Irregular menses were more common in those with a normal endometrium, while heavy menses predominated in those with an abnormal endometrium, with statistically significant differences (<0.001). The overall prevalence of obesity was 19.77%, but no clinical diseases were found significantly associated with abnormal endometrium. The prevalence of endometrial polyp and malignancy as confirmed by histopathology were 5.1% and 1.69%, respectively. No endometrial hyperplasia cases were reported.

CONCLUSION: Abnormal endometrial findings were relatively common among cases with polycystic ovaries, and most often, cases present with heavy menstrual bleeding. However, no predictable clinical factor can help identify PCOS patients with abnormal endometrial findings. Although malignancy was less common in the age group included in this study, the presence of abnormal sonographic findings would warrant further evaluation.

Keywords:

Endometrial pathology, international endometrial tumor analysis terminology, polycystic ovarian syndrome

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Background

Polycystic ovarian syndrome (PCOS) is the most common endocrine metabolic disorder among women, with a prevalence

of 3%–10% worldwide.^[1] It typically presents with menstrual irregularities such as amenorrhea and oligomenorrhea, alongside signs of hyperandrogenism, including hirsutism. Key metabolic features of PCOS include hyperinsulinemia, insulin

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resistance (IR), and obesity, which collectively increase the risk of type 2 diabetes mellitus and cardiovascular diseases.^[2]

Emerging evidence suggests that the endocrine and metabolic disruptions can significantly impact the endometrium in PCOS, leading to conditions like infertility, pregnancy complications, and endometrial disease.^[3] Dysregulated follicular development and chronic anovulation prevent regular shedding of the endometrium. Consequently, prolonged estrogen stimulation unopposed by progesterone leads to endometrial thickening, which can result in atypical hyperplasia and, in some cases, progress to endometrial cancer.^[4] Likewise, high androgen levels have been shown to induce endometrial hyperplasia and inhibit the growth and differentiation of endometrial cells in PCOS, reducing endometrial receptivity.^[3] One particular study has shown that 97% of a cohort of PCOS has anovulation, and 41% of them had endometrial hyperplasia.^[5]

Metabolic disturbances are also linked to endometrial abnormalities in patients with PCOS, with research showing that approximately 30% of those with endometrial lesions also experience IR.^[3] The abnormal endometrial microenvironment in PCOS patients with IR is responsible for the impaired endometrial receptivity, hyperplasia, and carcinogenesis. This may stem from multiple molecular mechanisms, including deficiencies in certain insulin receptor substrates,^[6] endometrial inflammation contributing to progesterone resistance,^[7] and an aberrant decidualization response to progesterone.^[8] Thus, endometrial findings in PCOS reflect a complex interplay of endocrine, metabolic, and inflammatory factors, which together contribute to different obstetric and gynecologic conditions.

The International Endometrial Tumor Analysis (IETA) group established a consensus that defined sonographic features for endometrial evaluation via gray-scale ultrasound, color Doppler, and sonohysterography, using standardized terms and definitions.^[9] Given the variability in endometrial sonographic features among women with PCOS, there is an opportunity to assess and refine the application of these findings that can help improve PCOS evaluation and management.

While studies have established the link of PCOS with malignant, pre- and nonmalignant endometrial lesions, local data on endometrial findings is limited. A local study has found a relatively increased prevalence of PCOS at 12.5% among premenopausal (30–39 years old) Filipino women diagnosed with endometrial cancer.^[10] So far, no local study has been conducted on the prevalence of abnormal endometrial findings among those with

PCOS, nor were these findings evaluated for association with clinical parameters, including the metabolic risk factors. This becomes significant to evaluate as racial and ethnic differences in the phenotype have been observed in women with PCOS.^[11–13] This study therefore aimed to evaluate the local prevalence and spectrum of endometrial findings among reproductive-aged women with PCOS in a tertiary hospital and correlate them with the clinical profile. Using IETA terminology, it explored the variety of endometrial appearances on sonography. Understanding these endometrial patterns may support improved management approaches for women with PCOS.

Methodology

Study design and study population

A 5-year retrospective review of cases was conducted at the Department of Obstetrics and Gynecology, University of the Philippines – Philippine General Hospital (UP-PGH). The UP Manila Research Ethics Board approved the study protocol. The study included all women aged 18–40 years old who had an ultrasound at the Division of Ultrasound, UP-PGH from January 2016 to December 2020 and with sonographic features of polycystic ovary or ovaries according to the Rotterdam sonographic diagnostic criteria, i.e., ≥ 12 follicles within the ovary with a diameter of 2–9 mm and/or ovarian volume $\geq 10 \text{ cm}^3$ involving one or both ovaries.^[14] All these women were also clinically diagnosed with PCOS based on the same criteria. Excluded were those with other ovarian nonmalignant abnormalities (such as endometriotic cysts and dermoid cysts), ovarian malignancy, pregnant patients, and patients diagnosed with congenital adrenal hyperplasia, Cushing's syndrome, and androgen-secreting tumors. Also excluded were those with incomplete charts and ultrasound reports.

Computation of sample size

The minimum required sample size was computed to be 107 patients,^[15] based on the 988 number of cases seen at the ultrasound unit with sonographic features of PCOS in a 5-year period (2016–2020), a 19.23% reported prevalence of abnormal endometrium,^[16] a 5% level of significance, and a 7.5% desired margin of error.

Conduct of the study

A total of 177 women were identified and included in the study based on the ultrasound reports obtained from the Division of Ultrasound. The demographic information and clinical data were obtained from the patient's charts. The ultrasound reports were re-evaluated by the investigators and verified based on the images and video clips. Descriptions used were

based on IETA terminologies.^[9] Those with proliferative phase, secretory phase, and thin endometrium in the final ultrasound impression were categorized as normal endometrium. Abnormal endometrium were those cases with ultrasound impressions of thickened endometrium, polyp, submucous myoma, and malignancy. The histopathologic results of patients who underwent surgery were reviewed and analyzed.

Statistical analysis

All the data were encoded and analyzed using the STATA 13.1 software (Stata Statistical Software: Release 13. College Station, TX: StataCorp LLC). Descriptive statistics were used to summarize the demographic and clinical characteristics of the patients. Frequency and proportion were used for categorical variables, the median and interquartile range (IQR) for nonnormally distributed continuous variables, and mean and standard deviation for normally distributed continuous variables. The odds ratio and corresponding 95% confidence intervals from binary logistic regression were computed to determine significant clinical factors associated with abnormal endometrial findings. All statistical tests were two tailed-test. Shapiro–Wilk was used to test the normality of the continuous variables. Missing values were neither replaced nor estimated. Null hypotheses were rejected at 0.05 α -level of significance.

Results

A total of 177 women who met the inclusion criteria were included in the study. The majority of them had normal endometrial findings ($n = 138$; 78%) however, 39 (22%) had abnormal findings consisting of thickened endometrium (14.7%), polyp (5.1%), submucous myoma (1.1%) and malignancy (1.1%). The endometrial findings based on ultrasound impressions are summarized in Table 1.

Demographic and clinical characteristics

A summary of the demographic and clinical characteristics of the patients is presented in Table 2. There was no

Table 1: Endometrial findings on ultrasound among cases with polycystic ovarian syndrome

Ultrasound impressions	Number of cases ($n=177$), n (%)
Normal endometrial findings	
Proliferative phase	50 (28.2)
Secretory phase	46 (26.0)
Thin endometrium	42 (23.7)
Abnormal endometrial findings	
Thickened endometrium	26 (14.7)
Polyp	9 (5.1)
Others	
Submucous myoma	2 (1.1)
Malignancy	2 (1.1)

significant difference between those with normal and abnormal sonologic endometrial findings in terms of age, body mass index (BMI), gravidity/parity, biochemical parameters (thyroid function tests), and associated diseases. For the biochemical parameters, the thyroid function tests were the only hormonal assays included in the clinical data obtained. Other relevant hormonal assays (luteinizing hormone [LH], total testosterone, free testosterone, insulin, sex hormone-binding globulin, follicle-stimulating hormone [FSH], estradiol [E2], dehydroepiandrosterone sulfate, and androstenedione) were not routinely requested.

Comparing the clinical presentation between the two groups, irregular or with missed menses (63.16%, $P = 0.039$) was common in the group with normal endometrium while prolonged bleeding was common in the group with abnormal findings (27.27%, <0.001).

Ultrasound findings using the international endometrial tumor analysis terminologies of patients with polycystic ovarian syndrome

Using the IETA descriptions, we identified the sonologic features that are common among the cases with and without abnormal endometrial findings [Table 3]. Patients with abnormal endometrial findings had significantly thickened endometrium (0.9; IQR = 0.7–1.4), nonuniform echogenicity (<0.001), and the presence of vascularity (<0.001). In contrast, those with normal findings had uniform echogenicity (either hyperechoic – 66.17% or trilaminar – 33.08%, <0.001) and linear endometrial midline (73.68%, <0.001). Most of the cases seen had regular endometrial myometrial junction (EMJ) regardless of the endometrial findings, if normal or abnormal. The absence of intracavitary fluid was significantly noted among those with normal endometrial findings ($P = 0.015$).

Histopathologic and sonologic endometrial findings

Only nine patients (5.1%) underwent surgical intervention [Table 2], and all had abnormal endometrial findings on ultrasound [Table 4]. The surgeries performed were endometrial biopsy, endometrial curettage, and hysteroscopic-guided endometrial curettage. There were 3 cases (1.69%) with a final histopathologic diagnosis of malignancy out of the 177 women with PCOS. The ultrasound findings/impression of these 3 cases were either thickened endometrium ($n = 1$) or malignancy ($n = 2$). The other remaining cases of thickened endometrium by ultrasound had benign or nonmalignant histopathologic results which included secretory phase endometrium and disordered proliferative endometrium. There were no cases of endometrial hyperplasia. The other ultrasound findings of

Table 2: Demographic and clinical characteristics of patients with polycystic ovarian syndrome according to endometrial findings

	Endometrial findings by ultrasound			P
	Total (n=177), frequency (%)	Abnormal (n=44; 25%), frequency (%)	Normal (n=133; 75%), frequency (%)	
Age, mean±SD	26.37±5.58	26.98±5.53	26.17±5.61	0.409
BMI	24.74±4.78	24.84±4.77	24.71±4.80	0.872
Normal	68 (38.42)	16 (36.36)	52 (39.1)	0.552
Overweight	39 (22.03)	8 (18.18)	31 (23.31)	
Preobese	35 (19.77)	12 (27.27)	23 (17.29)	
Obese	35 (19.77)	8 (18.18)	27 (20.30)	
Gravidity, median (IQR)	0 (0–1)	0 (0–1)	0 (0–1)	0.740
0	119 (67.23)	31 (70.45)	88 (66.17)	0.772
1	34 (19.21)	6 (13.64)	28 (21.05)	
2	14 (7.91)	4 (9.09)	10 (7.52)	
3	5 (2.82)	2 (4.55)	3 (2.26)	
4	3 (1.69)	1 (2.27)	2 (1.5)	
5	2 (1.13)	0	2 (1.5)	
Parity, median (IQR)	1 (1–2)	1 (1–2)	1 (1–2)	0.847
0	8 (13.79)	3 (23.08)	5 (11.11)	0.687
1	30 (51.72)	5 (38.46)	25 (55.56)	
2	13 (22.41)	3 (23.08)	10 (22.22)	
3	5 (8.62)	2 (15.38)	3 (6.67)	
4	1 (1.72)	0	1 (2.22)	
5	1 (1.72)	0	1 (2.22)	
Clinical presentation of PCOS				
Irregular w/missed menses	104 (58.76)	20 (45.45)	84 (63.16)	0.039
Acne	51 (28.81)	10 (22.73)	41 (30.83)	0.304
No menses	41 (23.16)	10 (22.73)	31 (23.31)	0.937
Prolonged menses	21 (11.86)	12 (27.27)	9 (6.77)	<0.001
Abnormal hair growth	8 (4.52)	2 (4.55)	6 (4.51)	0.992
Others	5 (2.82)	1 (2.27)	4 (3.01)	0.799
Biochemical parameter				
Thyroid-stimulating hormone	42 (23.73)	13 (29.55)	29 (21.8)	0.295
None	135 (76.27)	31 (70.45)	104 (78.2)	
Associated diseases				
Diabetes mellitus	8 (4.52)	1 (2.27)	7 (5.26)	0.408
Hypertension	4 (2.26)	1 (2.27)	3 (2.26)	0.995
Dyslipidemia	4 (2.26)	1 (2.27)	3 (2.26)	0.995
Metabolic diseases	3 (1.69)	1 (2.27)	2 (1.5)	0.732
Others	2 (1.13)	1 (2.27)	1 (0.75)	0.408
Surgery performed (total)				
Hysteroscopic	9 (5.08)	9 (5.08)	0	
Endometrial sampling	4 (2.26)	4 (9.09)	0	
Endometrial curettage	3 (1.69)	3 (6.82)	0	
Endometrial biopsy	2 (1.13)	2 (4.55)	0	
None	168 (94.92)	35 (79.55)	133 (100)	

SD: Standard deviation, IQR: interquartile range, PCOS: Polycystic ovarian syndrome, BMI: Body mass index

polyp (5.1%) and submucous myoma (1.1%) were confirmed on histopathology.

Discussion

Abnormal endometrial findings were found in this study to be relatively common among cases with PCOS, and most often these cases presented with heavy menstrual bleeding. Abnormal ultrasound findings based on the

IETA features that were significantly common include thickened endometrium, nonuniform echogenicity, and the presence of vascularity. Among the cases with ultrasound diagnosis of PCOS, only 5.1% had the benefit of histopathologic confirmation and a 1.69% overall prevalence of malignancy.

PCOS is characterized by IR, elevated LH levels leading to hyperandrogenism, and low FSH levels, which

Table 3: Ultrasound findings using the international endometrial tumor analysis terminologies among patients with polycystic ovarian syndrome

	Endometrial findings by ultrasound			P
	Total (n=177), frequency (%)	Abnormal (n=44; 25%), frequency (%)	Normal (n=133; 75%), frequency (%)	
Endometrial thickness, median (IQR)	0.7 (0.5–1)	0.9 (0.7–1.4)	0.6 (0.5–0.9)	<0.001
Uniform echogenicity				
Hyperechoic	98 (55.37)	10 (22.73)	88 (66.17)	<0.001
Trilaminar/three-layer pattern	48 (27.12)	4 (9.09)	44 (33.08)	
None	31 (17.51)	30 (68.18)	1 (0.75)	
Nonuniform echogenicity				
Homogeneous background	7 (3.95)	7 (15.91)	0	<0.001
Heterogeneous background	22 (12.43)	22 (50)	0	
None	148 (83.62)	14 (34.09)	133 (100)	
Endometrial midline				
Linear	103 (58.19)	5 (11.36)	98 (73.68)	<0.001
Nonlinear	2 (1.13)	1 (2.27)	1 (0.75)	
Irregular or not defined	1 (0.56)	1 (2.27)	0	
Not defined	53 (29.94)	31 (70.45)	22 (16.54)	
None	18 (10.17)	6 (13.64)	12 (9.02)	
Endometrial-myometrial junction				
Regular	173 (97.74)	43 (97.73)	130 (97.74)	0.685
Irregular	1 (0.56)	1 (2.27)	0	
Interrupted	1 (0.56)	0	1 (0.75)	
Not defined	1 (0.56)	0	1 (0.75)	
None	1 (0.56)	0	1 (0.75)	
Intra-cavitary fluid				
Absent	174 (98.31)	41 (93.18)	133 (100)	0.015
Low-level echogenicity	2 (1.13)	2 (4.55)	0	
Anechoic	1 (0.56)	1 (2.27)	0	
Endometrial outline				
Smooth	3 (1.69)	3 (6.82)	0	0.003
Endometrial folds	1 (0.56)	1 (2.27)	0	
None	173 (97.74)	40 (90.91)	133 (100)	
Intracavitary lesions				
Localized - sessile	2 (1.13)	2 (4.55)	0	0.061
None	175 (98.87)	42 (95.45)	133 (100)	
Color score (color and power Doppler)				
1	162 (91.53)	29 (65.91)	133 (100)	<0.001
2	10 (5.65)	10 (22.73)	0	
3	4 (2.26)	4 (9.09)	0	
4	1 (0.56)	1 (2.27)	0	
Vascular pattern				
Dominant vessel - single vessel	7 (3.95)	7 (15.91)	0	<0.001
Multiple vessels - multifocal origin	2 (1.13)	2 (4.55)	0	
Multiple vessels - focal origin	1 (0.56)	1 (2.27)	0	
Scattered	4 (2.26)	4 (9.09)	0	
Circular	2 (1.13)	2 (4.55)	0	
None	161 (90.96)	28 (63.64)	133 (100)	

SD: Standard deviation, IQR: interquartile range

often result in anovulation. Abnormal menstruation, a common presentation in PCOS, frequently necessitates sonographic evaluation to confirm PCOS and to evaluate the endometrium. In this study, a significant proportion of women with polycystic ovarian disease exhibited abnormal endometrial findings, including polyps, submucosal myomas, and thickened

endometrium – some even with endometrial cancer as confirmed on histopathology. These findings are consistent with the hormonal imbalance associated with the condition, in particular, elevated levels of estrogen without sufficient progesterone to balance it out. With infrequent menstruation, there are prolonged periods of unopposed estrogen exposure leading to thickening

Table 4: Endometrial impression on ultrasound and histopathology of nine (9) patients who underwent surgery

Endometrial impression on ultrasound	Case procedure	Histopathology of the endometrium
Malignancy	Endometrial biopsy	Malignancy
	Endometrial curettage	Malignancy
Thickened endometrium	Endometrial biopsy	Secretory phase
	Endometrial curettage	Disordered proliferative endometrium
	Hysteroscopic-guided endometrial curettage	Malignancy
	Endometrial curettage	Secretory phase
Submucous myoma	Hysteroscopic-guided endometrial curettage	Submucous myoma
	Hysteroscopic-guided endometrial curettage	Submucous myoma
Polyp	Hysteroscopic-guided endometrial curettage	Endometrial polyp

of the endometrial lining and making polyp formation more likely. Concurrently, without regular ovulation, progesterone production is reduced, which would normally help regulate and shed the endometrial lining. In addition, IR associated with increased levels of insulin and insulin-like growth factors, contributes further to stimulate endometrial growth and polyp formation.^[3] While polyps are usually benign, they can contribute to symptoms such as irregular bleeding, especially in women with already irregular cycles, and hence the need to monitor or even remove them if symptomatic. However, since PCOS is associated with a higher risk of both premalignant and malignant endometrial polyps in premenopausal women, it has been suggested that active management should be the approach regardless of the symptoms.^[17] The same underlying hormonal changes are responsible for the increased likelihood of developing endometrial hyperplastic changes and endometrial cancer.^[3] Interestingly, the prevalence of endometrial polyps and endometrial neoplasia observed here was low similar to some of previous reports,^[17,18] no case of endometrial hyperplasia was noted in this report, unlike some other published data which showed very high prevalence of hyperplasia.^[19] The results of the present study therefore seem to suggest that routine endometrial screening of all women with PCOS may not be recommended. However, a meta-analysis has shown that women with PCOS are up to 5 times more likely to develop endometrial cancer compared to those without PCOS, and the lifetime risk of endometrial cancer in patients with PCOS may be estimated to be as high as 12%–15%.^[20] Hence, a high index of suspicion for premalignant and malignant changes correlated with the clinical profile should still guide clinicians on the diagnostic management of such cases.

The development of submucous myomas is influenced by the hormones, estrogen and progesterone, however they are not directly associated with PCOS, similar to the way endometrial polyps are. Hormonal imbalance, in particular estrogen dominance together with obesity and IR are factors associated with both polyps and submucous myoma.^[21,22] Despite these overlaps, however, PCOS and myoma do not have a direct

causal relationship. Hence, the presence of submucous myoma in a woman with PCOS would generally be managed independently, based on factors related to the character of the myoma itself, including size, location, and associated symptoms.

While the link between chronic anovulation, abnormal endometrial pathology, and PCOS is well established, the characteristic endometrial appearance on imaging has received less attention. Based on the IETA description, the most common abnormal finding in this study was thickened endometrium, specifically for thickness >8 mm. This finding has been found to be a significant predictor of endometrial pathology in PCOS, regardless of patient age.^[21] While cut-off values for the reproductive age group remain uncertain, it has been found in one study that endometrial thickness of ≥ 8 mm was significantly associated with hyperplasia or carcinoma among those with perimenopausal bleeding.^[23]

The transitional region between the outer smooth muscle layer of the myometrium and the mucous membrane that makes up the endometrium is known as the EMJ. Also known as the junctional zone, it can appear altered in various uterine or endometrial diseases including adenomyosis, uterine fibroids, endometrial polyps, endometrial cancer, and congenital anomalies.^[24] However, it is not always abnormal even in the presence of endometrial pathologies, as demonstrated in this study. Focusing on the cases with histopathologic findings, only one case of endometrial cancer presented with irregular EMJ, and this was associated with high color score of 3–4. Irregular or disrupted EMJ is most commonly observed in more advanced cases of endometrial cancer, as tumor infiltration into the myometrium progresses. However, in early-stage or low-grade tumors, the EMJ appears relatively intact on imaging. Likewise, hyperplastic changes of the endometrium usually have thickened endometrium but with EMJ not necessarily disrupted, unless it has progressed to carcinoma. Benign lesions can also present with interrupted EMJ such as in cases of adenomyosis, where endometrial tissue grows into the myometrium, forming a thickened heterogeneous EMJ that appears indistinct on imaging. The vascular

pattern and echogenicity can help establish the diagnosis, especially in women with PCOS presenting with heavy menstrual bleeding. Both pathologies have heterogeneous echogenicity, but endometrial polyps typically present with regular EMJ with a single feeding vessel whereas carcinoma will have branching multifocal vessels. It is therefore important to note that while EMJ abnormalities as seen on ultrasound can suggest endometrial pathologies including carcinoma, it is not always abnormal in these conditions and may be seen in otherwise benign pathologies, and hence for cases with PCOS, they should be interpreted alongside other clinical findings and imaging features.

The link between PCOS and specific diseases or clinical factors is also well established. Women with PCOS have higher prevalence of hypertension, diabetes mellitus, dyslipidemia, obesity, and metabolic diseases.^[25,26] Interestingly, emerging studies have shown that metabolic disturbances are closely associated with endometrial abnormalities in patients with PCOS.^[3] It has been reported that approximately 30% of individuals with endometrial lesions also exhibit IR. Through various molecular mechanisms, IR results to altered endometrial microenvironment in PCOS which contributes to impaired endometrial receptivity, the development of hyperplasia, and an increased risk of carcinogenesis. Thus, endometrial findings in PCOS reflect a complex interplay of endocrine, metabolic, and inflammatory factors, that collectively cause these endometrial problems. This study therefore investigated the possible association of the abnormal endometrial findings of the PCOS cases with clinical factors including these diseases to identify predictable factors that may help in the management of PCOS. While no significant difference in the BMI was demonstrated between the 2 groups (normal and abnormal endometrium), the overall number of Filipino patients with obesity was high. However, only a handful of these PCOS patients had any of the associated diseases and metabolic problems, with no differences between those with normal and endometrial findings. Hence the current results do not point to any predictable clinical factor that may help identify PCOS patients with abnormal endometrial findings.

Compared with other populations, the disparity in the reported prevalence of the associated diseases may possibly be due to racial and ethnic differences, as they have been previously observed to influence the phenotype in women with PCOS.^[11-13] Asian women with PCOS have lower BMI compared with other ethnic groups, but East Asian women in particular had the highest prevalence for metabolic syndrome. In contrast, South Asians in particular have a high prevalence of IR and metabolic syndrome and are at risk for type 2 diabetes.^[13] Asian women with PCOS were also reported more likely to

have diabetes compared with Caucasian patients and thus may have risk for metabolic complications.^[12] While they are less likely to be obese, even at low BMI levels; however, they may have central adiposity which is a risk factor for metabolic complications.^[13] These differences in the ethnic groups studied are likely multifactorial in origin involving genetic, lifestyle, and environmental factors. Nevertheless, despite these differences, it is essential to monitor Asian women with PCOS including Filipino patients for metabolic risk, particularly due to central obesity and the predisposition to diabetes even at lower BMI.

Summary and limitations of the study

In summary, this study has shown that the overall prevalence of abnormal endometrial findings on ultrasound is relatively high, not necessarily consistent with previous reports. The overall prevalence of endometrial polyp and carcinoma are low while no endometrial hyperplasia cases were reported. The variability in reported prevalence may be attributed to the population included in the study. For instance, this study involved a cohort of PCOS cases diagnosed by ultrasound and clinical findings, and with only a few cases having confirmatory histopathologic diagnosis for the endometrium. This is unlike other studies which evaluated the histopathology of all cases undergoing surgical diagnostic evaluation, regardless of the ultrasound findings. Hence, small pathologies which may not necessarily be seen on routine/conventional ultrasound or would not warrant surgical intervention based on the clinical presentation may have been missed in this current study.

The variability in the results of PCOS research including the current study has been attributed not only to the differing populations studied, but the heterogeneity in the criteria used for the diagnosis of PCOS and the metabolic diseases. The cut-off values for diagnosis of the metabolic diseases may also be variable. Another significant limitation in this study is the absence of biochemical parameters which were not routinely requested due to financial constraints on the part of the patients. Likewise, other relevant laboratory tests (e.g. lipid profile and screening tests for diabetes) may not have been performed, potentially failing to obtain a complete diagnosis. Hence, for more conclusive reports, future studies may warrant a prospective design using standardized criteria and with ethnical differences taken into consideration.

Conclusion

Abnormal endometrial findings on ultrasound were relatively common among the cohort of PCOS studied, and most often, they presented with heavy menstrual

bleeding. However, there was a low prevalence of endometrial polyp and endometrial cancer, while no case of endometrial hyperplasia was reported. Significant ultrasound findings that consistently suggested the presence of abnormal endometrium included thickened endometrium, nonuniform echogenicity, and the presence of vascularity. Although malignancy was less common in the age group included in this study, the described abnormal sonographic findings would still warrant further evaluation.

The current results do not point to any predictable clinical factor that may help identify PCOS patients with abnormal endometrial findings. This is inconsistent with previous reports that have established close association between metabolic disturbances and endometrial abnormalities in patients with PCOS. Nevertheless, this report showed that the overall prevalence of obesity in the cohort was high. Hence, in the presence of central obesity and previous published reports on predisposition to diabetes even at low BMI among Asian women, patients with PCOS in the local population warrant monitoring for metabolic risk.

Authorship contributions

MKSZ, LDB, and MDA - Involved in the conceptualization and design of the study.

MKSZ - Involved in investigation (data collection), data curation and writing of the original draft; MKSZ, and MDA involved in formal analysis, and writing of the finalpaper.

LMDB - Investigation (reviewed ultrasound images), supervision.

MDA - Review, editing, and visualization of final work.

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Conflicts of interest

There are no conflicts of interest.

Data availability statement

The data that support the findings of this study are available on request from the corresponding author, MA.

References

- Wolf W, Wattick R, Kinkade O. Geographical prevalence of polycystic ovarian syndrome as determined by region and race/ethnicity. *Int J Environ Res Public Health* 2018;15:2589.
- Giudice LC. Endometrium in PCOS: Implantation and predisposition to endocrine CA. *Best Pract Res Clin Endocrinol Metab* 2006;20:235-44.
- Xue Z, Li J, Feng J, Han H, Zhao J, Zhang J, *et al.* Research progress on the mechanism between polycystic ovary syndrome and abnormal endometrium. *Front Physiol* 2021;12:788772.
- Dumesic DA, Lobo RA. Cancer risk and PCOS. *Steroids* 2013;78:782-5.
- Zhang H, Song X, Han Y, Xue F, Yang Z. Analysis of endometrial pathological status in patients with polycystic ovary syndrome. *Chin J Obstet Gynecol* 2007;42:493-4.
- Fornes R, Ormazabal P, Rosas C, Gabler F, Vantman D, Romero C, *et al.* Changes in the expression of insulin signaling pathway molecules in endometria from polycystic ovary syndrome women with or without hyperinsulinemia. *Mol Med* 2010;16:129-36.
- Patel BG, Rudnicki M, Yu J, Shu Y, Taylor RN. Progesterone resistance in endometriosis: Origins, consequences and interventions. *Acta Obstet Gynecol Scand* 2017;96:623-32.
- Piltonen TT, Chen JC, Khatun M, Kangasniemi M, Liakka A, Spitzer T, *et al.* Endometrial stromal fibroblasts from women with polycystic ovary syndrome have impaired progesterone-mediated decidualization, aberrant cytokine profiles and promote enhanced immune cell migration *in vitro*. *Hum Reprod* 2015;30:1203-15.
- Leone FP, Timmerman D, Bourne T, Valentin L, Epstein E, Goldstein SR, *et al.* Terms, definitions and measurements to describe the sonographic features of the endometrium and intrauterine lesions: A consensus opinion from the International Endometrial Tumor Analysis (IETA) group. *Ultrasound Obstet Gynecol* 2010;35:103-12.
- Ortega GM, Aguilar AS. Prevalence and characteristics of polycystic ovary syndrome (PCOS) in Filipino women diagnosed with endometrial cancer: A five-year retrospective study. *Philipp J Reprod Endocrinol Infertil* 2016;13:62-71.
- Engmann L, Jin S, Sun F, Legro RS, Polotsky AJ, Hansen KR, *et al.* Racial and ethnic differences in the polycystic ovary syndrome metabolic phenotype. *Am J Obstet Gynecol* 2017;216:493.e1-13.
- Kim JJ, Choi YM. Phenotype and genotype of polycystic ovary syndrome in Asia: Ethnic differences. *J Obstet Gynaecol Res* 2019;45:2330-7.
- Zhao Y, Qiao J. Ethnic differences in the phenotypic expression of polycystic ovary syndrome. *Steroids* 2013;78:755-60.
- Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. *Fertil Steril* 2004;81:19-25.
- Peacock JL, Peacock PJ. Research design. In: *Oxford Handbook of Medical Statistics*. United States: Oxford University Press; 2011. p. 60-1.
- Ignatov A, Ortmann O. Endocrine risk factors of endometrial cancer: Polycystic ovary syndrome, oral contraceptives, infertility, tamoxifen. *Cancers (Basel)* 2020;12:1766.
- Lu L, Luo J, Deng J, Huang C, Li C. Polycystic ovary syndrome is associated with a higher risk of premalignant and malignant endometrial polyps in premenopausal women: A retrospective study in a tertiary teaching hospital. *BMC Womens Health* 2023;23:127.
- Holm NS, Glintborg D, Andersen MS, Schledermann D, Ravn P. The prevalence of endometrial hyperplasia and endometrial cancer in women with polycystic ovary syndrome or hyperandrogenism. *Acta Obstet Gynecol Scand* 2012;91:1173-6.
- Park JC, Lim SY, Jang TK, Bae JG, Kim JJ, Rhee JH. Endometrial histology and predictable clinical factors for endometrial disease in women with polycystic ovary syndrome. *Clin Exp Reprod Med* 2011;38:42-6.
- Johnson J, Daley D, Tarta C, Stanciu PI. Risk of endometrial cancer in patients with polycystic ovarian syndrome: A meta-analysis. *Oncol Lett* 2023;25:168.
- Hou ZM, Sun Q, Liu YZ, Chen TF, Tang N. Effects of insulin resistance on myometrial growth. *Int J Clin Exp Med* 2015;8:1552-7.
- Parker WH. Etiology, symptomatology, and diagnosis of uterine myomas. *Fertil Steril* 2007;87:725-36.

23. Thoprasert P, Phaliwong P, Smachat B, Prommas S, Bhamarapratana K, Suwannarurk K. Endometrial thickness measurement as predictor of endometrial hyperplasia and cancer in perimenopausal uterine bleeding: Cross-sectional study. *Asian Pac J Cancer Prev* 2023;24:693-9.
24. Naftalin J, Jurkovic D. The endometrial-myometrial junction: A fresh look at a busy crossing. *Ultrasound Obstet Gynecol* 2009;34:1-11.
25. Lim SS, Davies MJ, Norman RJ, Moran LJ. Overweight, obesity and central obesity in women with polycystic ovary syndrome: A systematic review and meta-analysis. *Hum Reprod Update* 2012;18:618-37.
26. Sirmans SM, Pate KA. Epidemiology, diagnosis, and management of polycystic ovary syndrome. *Clin Epidemiol* 2013;6:1-13.