

Association of Serum Level of Bisphenol A with Polycystic Ovary Syndrome and Its Impact on Its Hormonal Profile

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ABSTRACT

Background: Bisphenol-A (BPA) may have a part in the aetiology of polycystic ovary syndrome (PCOS), or polycystic ovarian syndrome. BPA concentrations in biological fluids have been reported to be greater in PCOS women, and blood BPA levels in premenopausal women were associated with hepatic steatosis and indicators of low-grade inflammation.

Objective: This research aimed to determine the relationship between blood levels of Bisphenol-A, an endocrine disrupting substance (EDC), and PCOS as well as its effects on the hormonal profiles of the PCOS group in comparison to the healthy control group.

Subjects and methods: This cross-sectional study was carried out on 60 female participants who were selected from Outpatient Clinic of Obstetrics and Gynecology at Menoufia University Hospital and Damanhour National Medical Institute "The General Organization of Teaching Hospitals and Institutes through the period from August 2021 to January 2023. **Results:** The cutoff value of BPA sera levels in PCOS group was 12.50, with Sensitivity of 95% and Specificity of 85% at AUC of 0.590.

Conclusion: BPA sera levels was correlated with different gynecological complications like infertility, abnormal uterine bleeding (AUB), hirsutism and different metabolic complications like DM and chronic HTN as a secondary outcome in PCOS group.

Keywords: BPA, Hormonal profile, Infertility, PCOS.

INTRODUCTION

The most prevalent endocrinopathy in premenopausal women is PCOS. Despite the fact that the aetiology of PCOS already encompasses a complex combination of genetic and epigenetic variables, medical and scientific understanding of the issue is still insufficient [1, 2].

The presence of any or all of the following symptoms, including irregular menstrual cycles (amenorrhea or oligomenorrhea), an excess of androgen, anovulation that result in infertility, and polycystic ovaries as seen on ultrasound, is required to diagnose this heterogeneous disease [3]. Additionally, PCOS sufferers experience higher rates of weight gain and obesity prevalence, which makes the problem more severe, causes great concern for people who are affected, and necessitates attention to a healthy lifestyle [4]. PCOS has the potential to have detrimental effects, such as an increased risk of endometrial hyperplasia and neoplasia [5].

It has been demonstrated that environmental endocrine disruptors (EEDs) can alter how the hypothalamic-pituitary-ovarian axis regulates hormones. EEDs are therefore intended to function as steroid-agonists and/or antagonists. Previous research also suggested a link between PCOS and prior EED exposure, as well as with alterations in environment and way of life [6].

One of the most prevalent plasticizers is BPA, which may be found in a number of everyday items such as food packaging, cans, electronics, dental sealant materials, carbonless receipts, eyeglasses, and

water pipes [8]. BPA is a substance known as an endocrine disruptor chemical (EDC) because of its estrogenic qualities, which indicates that it alters functions of the endocrine system and consequently causes adverse effects in an intact organism, or its progeny, or (sub)populations [9]. It has been shown and extensively discussed that BPA affects a wide range of cells both through conventional signaling (oestrogen receptors: ER and ER) and through unconventional pathways [10].

Furthermore, current research has emphasised its possible contribution to the aetiology of PCOS [11]. The gonadotropin-releasing hormone (GnRH) pulse generator may be activated by hypothalamic BPA exposure, which may then enhance luteinizing hormone (LH) and reduce follicle-stimulating hormone (FSH) production by the pituitary and induce ovarian hyperandrogenism [12].

Additionally, androgen synthesis in ovarian theca cells may be directly stimulated by BPA, resulting in hyperandrogenemia and consequent hyper-estrogenemia. BPA concentrations in biological fluids have been discovered to be greater in PCOS women, and blood BPA levels in premenopausal women have been linked to hepatic steatosis and indicators of low-grade inflammation [13]. Therefore, the goal of our investigation was to determine whether there was a relationship between serum BPA concentrations as an EDC and PCOS as well as how it affected the hormonal profile of the PCOS group in comparison with the healthy control group.

SUBJECTS AND METHODS

Study design and grouping: This cross-section study was carried out on 60 female participants who were selected from outpatient clinic of obstetrics and gynecology at Menoufia University Hospital and Damanhour National Medical Institute “The General Organization of Teaching Hospitals and Institutes” through the period from August 2021 to January 2023. The 60 participants were classified into two groups: **Group I (PCO group):** Consisted of 30 PCOS cases aged 17-40 years old diagnosed according to Rotterdam criteria. Then the patients were classified into subgroups, according to PCO phenotypes [4], [Phenotype-A (HA+OD+PCO), Phenotype-B (HA+OD) and Phenotype-C (HA+PCOM)]. **Group II (Control group):** Consisted of 30 healthy participants not receiving any hormonal contraceptives.

$$n \geq \left(\frac{Z_{1-\alpha/2} + Z_{1-\beta}}{\frac{1}{2} \log_e \frac{1+r}{1-r}} \right)^2 + 3$$

Sample size estimation: Based on previous study [14] who found that the mean serum BPA level was greater in women with PCOS, and there was a positive correlation between it with testosterone ($r=0.53$, $p=0.05$). Minimum sample size calculated for this study was 52 females according to the following formula:

Where, n : sample size, $Z_{1-\alpha}$: z score for CI 95% and equals 1.96, $Z_{1-\beta}$: z score for power of the study 80% and equals 0.84 r : correlation coefficient ($r=0.53$), A total number of 8 females will be added to calculated sample as drop-out rate (15% of calculated sample size)= 60 participants.

Inclusion criteria: According to the Rotterdam criteria [15], PCOS sufferers have to meet two of the following three criteria in addition to being excluded from other aetiologies such as congenital adrenal hyperplasia, Cushing syndrome, and androgen-secreting tumour, clinical and/or biochemical hyperandrogenism, as well as polycystic ovarian morphology on ultrasound (at least 20 follicles with a diameter of 2 to 9 mm and/or an ovarian volume of less than 10 mL in at least one ovary), are all indicators of this condition.

Exclusion criteria: As females age below 17 years old or above 40 years old, females receiving hormonal contraceptives, patients with endocrine diseases, patients with endometriosis & uterine fibroids, history of diabetes mellitus, history of cardiovascular disease, blood pressure more than 140/80 mmHg, renal failure, neoplastic disorders, and smoking.

Methodology: All patients participated in the study were subjected to the following:

- A detailed history taking including residency, level of education, career, marital status and parity,

menstrual history (oligomenorrhea, amenorrhea, age of menarche ...etc), medical history of any systemic disease (diabetes mellitus, thyroid disease, heart disease and endometrial cancer), drug history for any medications (types, duration & doses), family history of any similar condition.

- Through specific questionnaire we assessed the exposure to potential different sources of Bisphenol A (e.g. plastic food containers, plastic water bottles, food cans, white dental sealants, DVDs, CDs and thermal papers) among participants of the studied groups and the degree of exposure recorded and gives a score as follow: score 0 indicates no exposure, score 1-4 indicates minimal exposure, score 5-10 indicates moderate exposure and score 11-17 indicates severe exposure.
- General and local examination: General examination included vital signs (pulse blood pressure temperature), weight, height and BMI, bilateral breast examination for galactorrhea, thyroid gland examination and assessment of distribution and the degree of cutaneous manifestation of androgen excess (hirsutism, acne, and alopecia). Local examination included local abdominal examination in the form of inspection, superficial and deep palpation, percussion and auscultation and local pelvic examination in the form of inspection, digital PV, bimanual pelvic examination as well as speculum examination.
- Laboratory investigations: Serum level of the following in the follicular phase of the menstrual cycle (third day): LH/FSH ratio, free testosterone, 17-hydroxy-progesterone, androstenedione, sex hormone-binding globulin (SHBG) and serum BPA level. To rule out hypothyroidism, hyperprolactinemia, and non-classical congenital adrenal hyperplasia, respectively, measurements of TSH, prolactin, and 17-OH progesterone were taken.
- Transvaginal ultrasound in lithotomy position to all participated women using (Vinnox2 vaginal probe F4-9E, P0152GC059, China) with transducer frequency ≥ 8 MHZ.

The study outcomes:

Primary outcome are the correlation and association between serum levels of BPA and PCOS. Secondary outcomes include impact of serum BPA levels on the score of specific questionnaires used for assessment of exposure to potential different sources for BPA, hormonal profiles of PCOS, phenotypes of PCO and different gynecological and metabolic complications of PCOS.

Ethical committee:

The Ethics Committee of Menoufia Faculty of Medicine and Damanhour National Medical Institute granted the study approval. All participants signed informed consents after a thorough explanation of the goals of the study. The Helsinki Declaration was followed throughout the study's conduct.

Statistical analysis: Results were tabulated and statistically analysed using industry-standard software for Microsoft Windows 10 (MICROSOFT EXCEL 2019 and SPSS V. 25). There were two different types of statistics used: frequency and percentage for qualitative data and descriptive statistics, which comprise the description of the data in the form of mean \pm SD for quantitative data. One-way ANOVA (F) is an analytical statistic that uses Chi-Squared (χ^2) and the standard student t test (t). A significant level is defined as a $p \leq 0.05$.

RESULTS

Basal characteristics; menstrual pattern and degree of hirsutism using MFGS score: There were no

significant differences among polycystic ovary syndrome cases and control group regarding age, BMI, residency, parity, and bleeding duration ($P > 0.05$). While the number of highly educated patients and with career among PCOS group were 66.7%, 60% respectively. Also, the number of married patients were more among PCOS group (100%), ($P < 0.05$). In addition, the age of menarche ≥ 14 years, irregular menstruation and menstrual cycle length > 35 days were in PCOS group [50, 93.3 and 93.3 % respectively, ($P < 0.05$)]. Also, the number of moderate and severe degree of hirsutism among PCOS group had higher significant difference ($P < 0.001$) (**Table 1**).

Table (1): Basal characteristics, menstrual pattern and degree of Hirsutism using MFGS score of participants of both studied groups.

Variables	PCOS (n=30)		Control (n=30)		t	P value	
Age/ Year (Mean \pm SD)	29.00 \pm 6.96		29.43 \pm 6.62		0.247	0.806	
BMI(Kg/m ²) Mean \pm SD	22.63 \pm 1.22		22.77 \pm 1.30		0.433	0.667	
	No.	%	No.	%	χ^2		
Residency							
Rural	17	56.67	17	56.67	0.00	1.00	
Urban	13	43.33	13	43.33			
Education							
Low	3	10	15	50	11.667	0.003*	
Mid	7	23.3	5	16.7			
High	20	66.7	10	33.3			
Career							
Employed	18	60	10	33.3	4.286	0.038*	
Non-employed	12	40	20	66.7			
Marital status							
Single	0	0	5	16.7			
Married	30	100	25	83.3	5.455	0.020*	
Married	0	0	0	0			
Divorced	0	0	0	0			
Widow							
Parity				33.3			
Nullipara	15	50	10	66.7	1.714	0.190	
Multipara	15	50	20				
Age of menarche/years	10-13	15	50	30	100	20.000	<0.001*
	≥ 14	15	50	0	0		
Menstrual regularity							
Regular	2	6.7	30	100	52.500	<0.001*	
Irregular	28	93.3	0	0			
Menstrual cycle length/days							
<21	0	0	0	0	52.500	<0.001*	
21-35	2	6.7	30	100			
>35	28	93.3	0	0			
Bleeding duration/days					---	---	
<3	0	0	0	0			
3-8	30	100	30	100			
>8	0	0	0	0			
Degree of hirsutism							
No score (0)	5	16.67	19	63.33			
Mild hirsutism score (10-14)	9	30.00	9	30.00	19.500	<0.001*	
Moderate hirsutism score (15-24)	10	33.33	2	6.67			
Severe hirsutism (>24)	6	20.00	0	0.00			

χ^2 : Chi-square test. F: Standard student – t test *: Significant.

Hormonal characteristics in participants of both studied groups: There were no significant differences among polycystic ovary syndrome cases and control group regarding prolactin and 17-OH progesterone ($P > 0.05$). TSH, LH/FSH ratio, free testosterone, androstenedione were significantly higher among PCOS cases group (2.20 ± 0.63 , 3.05 ± 0.51 , 8.95 ± 3.07) than in control group (1.34 ± 0.36 , 1.26 ± 0.25 , 2.70 ± 0.50) respectively, ($P < 0.05$). On the other hand, SHBG was significantly less among polycystic ovary syndrome cases (12.57 ± 2.69) than in control group (56.97 ± 10.28), ($P < 0.05$) (**Table 2**).

Table (2): Hormonal characteristics in participants of both studied groups.

Variables	PCOS (n=30)	Control (n=30)	t	P-value
Prolactin (ng/ml) Mean \pm SD	20.17 \pm 4.91	22.95 \pm 5.64	0.647	0.750
17OH.Progesterone (ng/ml) Mean \pm SD	1.54 \pm 0.25	1.99 \pm 0.42	1.105	0.074
TSH (n mol/L) Mean \pm SD	2.31 \pm 0.23	2.19 \pm 0.23	2.054	0.044*
LH / FSH Ratio Mean \pm SD	2.20 \pm 0.63	1.34 \pm 0.33	2.000	<0.001*
Free testosterone (n. mol /l) Mean \pm SD	3.05 \pm 0.51	1.26 \pm 0.25	8.867	<0.001*
Androstenedione (n. mol/L) Mean \pm SD	8.95 \pm 2.21	2.70 \pm 0.50	12.188	<0.001*
SHBG (n mol/L) Mean \pm SD	12.57 \pm 2.69	56.97 \pm 10.28	1.448	<0.001*

Dietary & non-dietary BPA exposure among participants using specific questionnaire score: According to dietary & non-dietary BPA exposure, between the two investigated groups, there was a statistically significant difference, the PCOS group had the highest difference ($P < 0.001$) (**Table 3**).

Table (3): Dietary & non-dietary BPA exposure among participants using specific questionnaire score

BPA Exposure	PCOS (n=30)		Control (n=30)		X ²	P value
	N	%	N	%		
No exposure Score (0)	3	10	23	76.7	33.437	<0.001*
Minimal exposure Score (1-4)	4	13.3	5	16.7		
Moderate exposure Score (5-10)	15	50	2	6.7		
Severe exposure Score (11-17)	8	26.7	0	0		

X²: Chi square, *: Significant

Correlation of different BPA sera levels in both studied groups:

There was a statistically significant difference between the two analysed groups, as indicated by the association of various BPA sera levels in both study groups as 36.67% of PCOS patients who had level of 10-15.9 ng/ml vs 76.67% of controls. Also, 63.33% of patients had level of 16-25 ng/ml vs 13.33% only in controls [with ($P=0.025$, and 0.004 , respectively)] (**Figure 1 & table 4**).

Table (4): Correlation of different BPA sera levels in both studied groups

Different BPA serum levels (ng/ml)	PCOS (N=30)		Control (N=30)		Sig. test	P-value
	N	%	N	%		
Level (4 – 9.9)	0	0.00	3	10.00	NA	----
Level (10 – 15.9)	11	36.67	23	76.67	$\chi^2 = 3.67$	0.025*
Level (16 – 25)	19	63.33	4	13.33	$\chi^2 = 5.37$	0.004*
Mean ± SD	17.47 ± 3.50		12.37 ± 3.06		t= 5.203	< 0.001*

NA: not applicable

Correlation of different BPA Sera levels and impact on hormonal profile in PCOS group: There was significant relation between different BPA sera levels and impact on hormonal profile in PCOS group. Different BPA serum levels had more impact on androstenedione and SHBG (P<0.001) (Table 5).

Table (5): Correlation of different BPA serum levels and impact on hormonal profile in PCOS group

Different BPA serum levels (ng/ml)	LH/FSH ratio Mean± SD	Free testosterone Mean± SD	Androstenedione Mean± SD	SHBG Mean± SD
Level (4 – 9.9)	0	0	0	0
Level (10 – 15.9)	0.81 ± 0.20	0.97 ± 0.23	3.22 ± 0.80	4.60 ± 1.1
Level (16 – 25)	1.39 ± 0.33	1.7 ± 0.41	5.72 ± 1.41	7.76 ± 1.93
P value	0.036	0.025	0.001	0.001

NA: not applicable.

Cutoff value of BPA Sera Levels in PCOS group: The cutoff value of BPA sera levels in PCOS group was 12.50, with sensitivity of 95% and specificity of 85% at AUC of 0.590 (Figure 2).

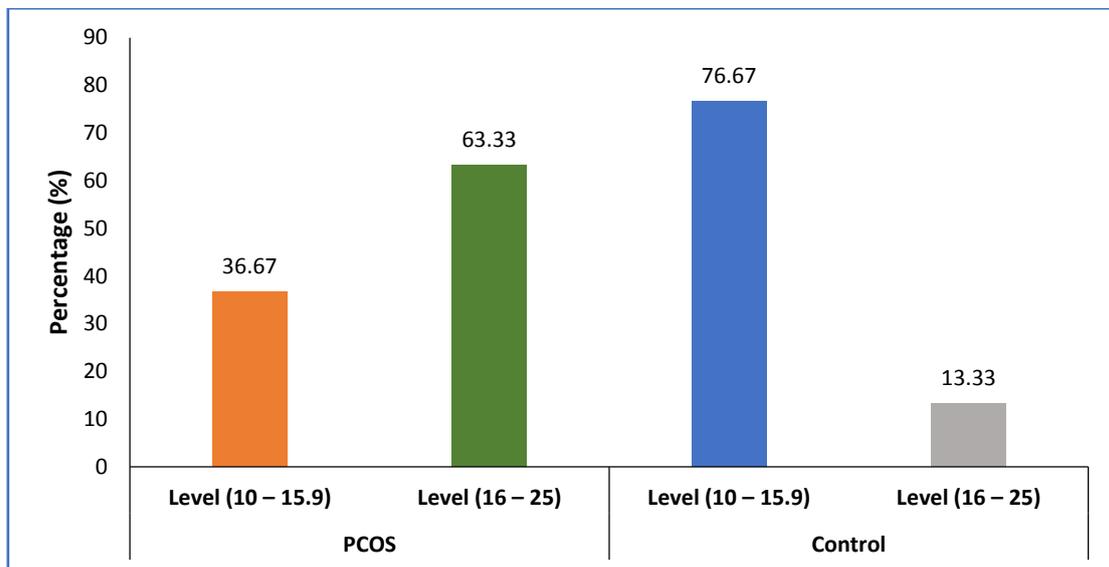


Figure (1): Correlation of different BPA serum levels in both studied groups.

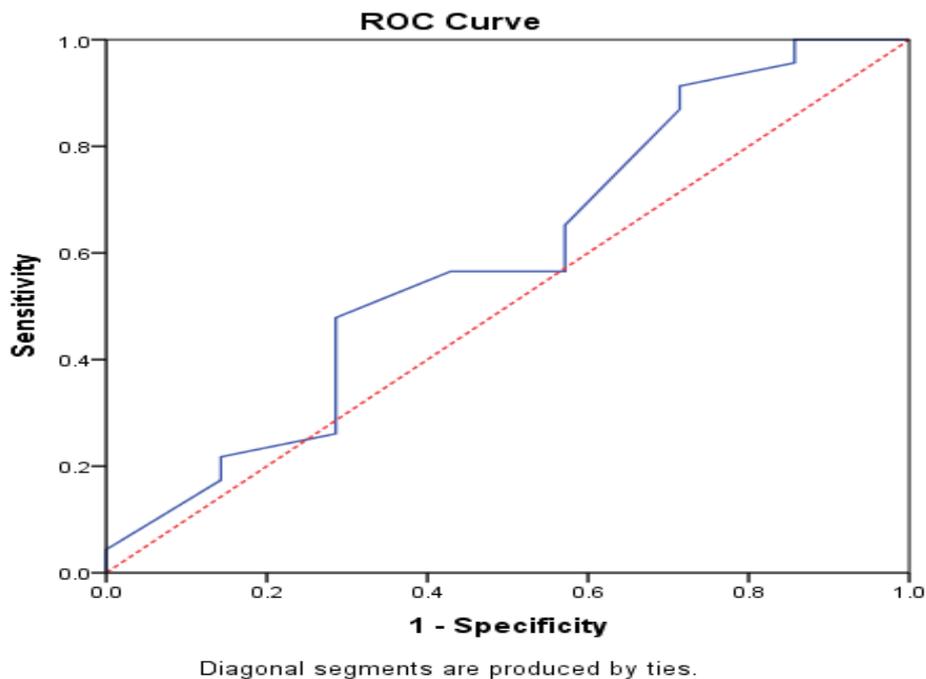


Figure (2): ROC curve to detect sera levels of Bisphenol A in PCOS group.

DISCUSSION

Our study's findings showed that there was a statistically significant difference between the two groups that had been examined for certain BPA sera levels as 36.67% of PCOs patients had level (10-15.9) vs 76.67% of controls, also, 63.33% of patients had level (16-25) vs 13.33% only in the controls with significance difference ($P=0.025$, and 0.004 , respectively). Also, our study showed that there was positive correlation between different BPA sera levels and serum androgens as there was significant increase in free testosterone and androstenedione among PCOS group (3.05 ± 0.51 , 8.95 ± 3.07) than the control group (1.26 ± 0.25 & 2.70 ± 0.50) respectively. In line with our study, **Takeuchi and colleagues** [16] revealed gender-specific variations in serum BPA concentrations. The blood BPA concentrations in this study's normal males and PCOS groups were both considerably greater than those in the normal women group, confirming the idea that BPA may be related to higher levels of androgen. BPA levels were surprisingly higher in PCOS patients as well as in healthy obese persons, indicating that PCOS and obesity are two distinct manifestations of the same metabolic illness [17]. In another investigation, the danger of estrogenic endocrine-disrupting substances on human reproduction was examined. The study's findings showed that, as compared to women without PCOS, those with PCOS had considerably higher serum BPA concentrations [18].

Additionally, in comparison with control group, **Kandaraki et al.** [19] assessed the levels of BPA in PCOS women as well as the relationship between

BPA and hormonal and metabolic markers. Compared to the control group, they discovered that PCOS women had greater BPA levels. Additionally, women with PCOS showed a statistically significant positive connection between androgens and BPA. According to **Tarantino et al.** [13], women with PCOS who have high blood BPA levels also have greater levels of inflammation, hepatic steatosis, free-androgen index (FAI), and insulin resistance. However, the research team reported a negative association between urinary BPA (U-BPA) levels and total testosterone, free testosterone, and FAI, suggesting contentious results regarding BPA and androgen levels. **Lazrová et al.** [20] found analogous results regarding high BPA levels and PCOS associations. Similar patterns can be seen in individuals with PCOS who had higher BPA concentrations in both urine and blood tests. BPA levels were found to be considerably higher in the PCOS group in a subset of Iranian women when compared to the control group ($p < 0.001$), suggesting a link between BPA exposure and PCOS [21]. Additionally, women with PCO had substantially higher urine BPA levels in a research by **Fathy et al.** [22] conducted in Egypt on December 20, 2022 as compared to women in the control group ($3439.05 \pm 2118.25 \mu\text{g} / \text{mL}$ vs. $813.84 \pm 584.787 \mu\text{g} / \text{mL}$ respectively, $P < 0.001$). Additionally, women in the PCO group had greater serum free testosterone levels than women in the control group ($4.71 \pm 0.98 \text{ pg/mL}$ vs. $2.23 \pm 0.65 \text{ pg/mL}$ respectively, $P < 0.001$).

Our study showed that PCOS group according to specific questionnaire was more exposed in daily life to dietary and non-dietary sources of BPA ($P < 0.001$)

like fast and canned food consumption habit, food packages and cans, bad quality plastic bottles, heating food in microwave, washing the plastic food containers in a dishwasher and presence of white dental sealant materials, which were correlated with increased sera BPA levels in PCOS group.

Concerning the impact of BPA on other hormones, our study showed that there were no significant differences among PCOS, and control group regarding prolactin and 17-OH progesterone ($P > 0.05$). While, TSH, LH/FSH ratio, were significantly higher among PCOS group (2.31 ± 0.23 & 2.2 ± 0.63) than in control group (2.19 ± 0.23 & 1.34 ± 0.36) respectively, ($P < 0.05$). In the opposite hand, SHBG was significantly lower among PCOS group (12.57 ± 2.69) than in control group (56.97 ± 10.28), ($P < 0.05$). In line with our current study in relation to LH/FSH ratio, a study on 2021 in Iraqi [23] showed that serum LH levels showed significant difference in patients group when compared to control. Other results of this study showed non-significant difference in serum FSH level was observed in patients group when compared with control. Regarding LH/FSH ratio, the results recorded a highly significant elevation in patients group when compared to control and have shown a highly significant direct correlation of serum BPA levels with LH/FSH ratio at a highly significant level. Other study explained the role of ratio in PCOS etiology and confirmed the role of BPA in pathophysiology of PCOS [24]. According to another study, polycystic ovarian syndrome-diagnosed infertile women's third day FSH levels are negatively correlated with BPA exposure [25].

Last but not least, research conducted in Egypt in December 2022 by **Fathy et al.** [22] revealed that women with PCOS had serum FSH levels that were considerably lower than those in the control group (5.14 ± 1.88 mIU/mL vs. 6.77 ± 1.52 mIU/mL, respectively, $P < 0.001$). Additionally, as compared to the women in the control group, the women with PCOS had a significantly higher level of serum LH (6.98 ± 2.14 mIU/mL vs. 3.61 ± 1.36 mIU/mL, respectively, $P < 0.001$). In a research similar to ours, **Bizo et al.** [26] discovered that the four PCOS phenotypes and BMI value were related to a decrease in SHBG concentration in the serum of women with PCOS. Previous cross-sectional research on adult patients found a negative correlation between blood TSH concentration and body fluid BPA level [27-31].

Our study showed that, the majority of patients (80%) of polycystic ovary syndrome had the phenotype (A). Our findings are consistent with those of **Sachdeva et al.** [32] who discovered that full-blown PCOS (phenotype A) was the most prevalent phenotype with a frequency of 67.7%, followed by phenotypes C, B, and D, in that order. However, **Elasam et al.** [33] discovered that among infertile Sudanese women, phenotype D was the most common (51.6%), followed by phenotype B (22.6%), phenotype C (18.2%), and

phenotype A (7.6%). These results are consistent with research by **Mehrabian et al.** [34] from China and Iran, which found that phenotype D was the most common.

The current study revealed that there were no significant differences among PCOS, and control group regarding age, body mass index, residency, parity and menstrual bleeding duration ($P > 0.05$). While the numbers of high educated patients and with a career among PCOS group were 66.7% & 60% respectively. Also, the number of married patients were more among PCOS group (100%) ($P < 0.05$) and the age of menarche ≥ 14 years, irregular menstruation and menstrual cycle length > 35 days were in PCOS group (50, 93.3 and 93.3 %) respectively, ($P < 0.05$). A study by **Elasam et al.** [33] found that the sample's median age was 26.0 ± 7.9 years, and 156 (42.4%) of the 274 women had jobs. In addition, the sample's median (interquartile range) gender was female. 156 (42.4%) of the women who had a physical examination were obese and 10 (2.7%) had hypertension. Another research by **Mohammed et al.** [35] found that infertile PCOS patients in Khartoum, Sudan had a substantially higher BMI than the general population.

Finally in our study by using ROC curve to detect sera levels of BPA in PCOS group, we showed that the cut off value of BPA sera levels was 12.50 at AUC of 0.59, which denoted that sera BPA levels had sensitivity of 95% and specificity of 85% that denoted the BPA important environmental factor in PCOS pathogenesis.

STRENGTHS OF THE STUDY

- The main strength of our study is the use of strict inclusion and exclusion criteria for choosing patients with PCOS and control groups. Women with endocrine problems, diabetes mellitus, HTN, cardiovascular diseases, neoplastic disorders and receiving any hormonal contraceptives were excluded.
- Each participant of both studied groups was subjected to specific questionnaire for dietary and non-dietary exposure to BPA to demonstrate environmental sources for BPA and assess the degree of its exposure.

LIMITATIONS OF THE STUDY

- The number of women involved in the current study was a constraint, and more women are needed to be included in future studies if the results of this study are to be supported by findings from other studies. Additionally, to demonstrate that either BPA is a cause of PCOS or that it is a result of its relationship with PCOS. The real exposure to this EDC may not always be reflected by serum BPA level.
- BPA was demonstrated to be rapidly metabolised in the liver and to have a half-life of several hours when it is present in blood. Some studies contend

that urine may be a better material to assess the exposure risk.

- The case-control of the current investigation was designed after PCOS was identified. Therefore, it was unable to draw a connection between BPA and PCOS. It is feasible to hypothesise that PCOS patients may have engaged in certain behaviours that made them more vulnerable to be exposed to BPA, or a reverse causation. Although this is a possibility, we were unable to come up with any solid arguments to back up this assumption. Additionally, people could exercise more caution when choosing what they consume and use, so minimising their chances of exposure. It's true that the best way to ascertain the connection between BPA and the likelihood of PCOS is through a prospective cohort research. However, carrying out such a study would be expensive and time-consuming.

CONCLUSION

Our research revealed that blood BPA concentration in PCOS-affected women are considerably greater than those in age- and BMI-matched controls. It also revealed that serum LH/FSH ratio, free testosterone, and androstenedione concentrations were favourably and SHBG adversely linked with each other. Therefore, our data not only supports the hypothesis that BPA may be a possible environmental component contributing to the development of PCOS but also suggests that BPA might affect the hormonal profile in PCOS women.

Our study showed that PCOS group according to specific questionnaire was more exposed in daily life to dietary and non-dietary sources of BPA like fast and canned food consumption habit, food packages, bad quality plastic bottles, heating food in microwave, washing the plastic food containers a dishwasher and presence of white dental sealant materials. It showed that BPA can be a possible environmental component contributing to the development of PCOS.

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Competing interests: Nil.

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