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Biochemistry

ASSOCIATION OF PROSTATE SPECIFIC ANTIGEN IN POLYCYSTIC OVARY SYNDROME

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ABSTRACT **Background:** Prostate specific antigen (PSA) has been observed to increase in polycystic ovary syndrome (PCOS). Expression of prostate specific antigen (PSA) gene is thought to be under androgenic regulation. Therefore, PCOS, a hyperandrogenic state is expected to be associated with higher level of PSA. **Aims:** In the current study, we aimed to determine levels of PSA in PCOS patients and healthy controls, correlation between PSA and hirsutism and androgen levels and diagnostic value of PSA level in PCOS. **Materials And Methods:** A total of 50 women with PCOS and 50 aged matched healthy females were recruited in this case-control study. The subjects were compared by means of serum PSA level. 50 PCOS patients diagnosed on the basis of revised Rotterdam 2003 criteria. Serum PSA (ng/ml) and testosterone (ng/dl) were measured along with ultrasonogram (USG) for pelvic organs. PSA and testosterone was measured by direct chemiluminometric technology. **Results:** Serum PSA and testosterone level were significantly higher in PCOS subjects than control (0.05±0.01 vs. 0.04±0.01ng/ml; p<0.02; 40.18±21.12 vs. 28.92±13.65ng/ dl; p<0.01 respectively). PSA positively correlated with observed polycystic ovaries by USG in the PCOS group (r=0.397, p=0.011) but not with testosterone (r= 0.075, p=0.644). **Conclusion:** Present study observed that serum PSA is higher in PCOS patients than control and correlate with polycystic ovary detected by USG.

KEYWORDS : PCOS, PSA, testosterone.

INTRODUCTION:

Polycystic ovary syndrome (PCOS), one of the most frequent endocrine disorders in reproductive aged women, is a syndrome of ovarian dysfunction. 4-11% of women of reproductive age group in Indian population suffer from Polycystic Ovarian Syndrome(PCOS), which is the most common endocrine reproductive disorder of Females.[1] PCOS is an important cause of both menstrual irregularity and androgen excess in women. When fully expressed, the manifestations include irregular menstrual cycles together with hirsutism and/or acne; obesity is a frequent concomitant.[2] Androgen excess is the main pathology in PCOS. Insulin resistance with the consequent hyperinsulinemia has a pathogenic role in PCOS.[3]Insulin can promote ovarian androgen production, either directly by stimulating ovarian enzyme complex P450c17α or indirectly by stimulating pituitary luteinizing hormone (LH) secretion.4In vitro studies have also suggested amplification of ACTH (Adrenocorticotropic hormone) stimulated adrenal androgen secretion by hyperinsulinemia.[4]

Prostate specific antigen (PSA) is determined to be a 33-kDa serine protease that is primarily a product of prostatic tissue and secreted into the seminal fluid.[4] PSA is used as a highly specific and valuable marker of prostatic carcinoma regarding the screening, diagnosis and monitoring of disease.[5] Recent development of ultrasensitive assays demonstrated presence of PSA in wide variety of female tissues and fluids particularly breast, ovary, milk, endometrium and amniotic fluid.[6] However, the main source of serum PSA in women is still unknown, although some authors suggest that it is produced by periurethral glands which are highly homologous to male prostate. The gene expression and protein production of PSA in non prostatic tissues are under the regulation of steroid hormones via their receptors. Androgens, glucocorticoids and progestins regulate the PSA gene expression. As a diagnostic marker of PCOS, PSA level has been found to have relatively high specificity (80%) and sensitivity (70%).[7] Serum PSA level remain stable throughout the menstrual cycle with less intracyclic variations.[8] PSA is not operator dependent, like ultrasound ovarian morphology and also widely available. Hence, PSA value could be a diagnostic marker of PCOS. To assess whether serum PSA assay may fulfill this role, the present study was designed to see the serum PSA level in PCOS women and control subjects in a tertiary level hospital.

METHODS:

This study was a case-control study conducted in Department of Biochemistry, North Bengal Medical College in collaboration with Department of Obstetrics and Gynaecology in a period of 1 year, 2024-2025. All patients were 20-35 years old and referred for oligomenorrhea and hirsutism to our outpatient clinic. The exclusion criteria were having adrenal enzyme defects, androgen secreting adrenal and ovarian tumors, Cushing's syndrome, hyperprolactinemia, thyroid dysfunction and idiopathic hirsutism. Patients who had been treated for PCOS previously were excluded too. Control women were eumenorrheic, non-hirsute, without any family history of hirsutism and had no endocrine disorders. Women who did not agree to participate in the study or detected pregnant were excluded. This study was approved by the Institutional Review Board of the hospital. Informed written consent was obtained from PCOS patients as well as from controls. PCOS patients were diagnosed on the basis of Revised Rotterdam Consensus 2003 criteria.12 Samples for hormonal assay were taken on the early follicular phase (days 3-5 of menstrual cycle) in women with regular menstrual cycle or oligomenorrhoea and on any other day in patients with amenorrhea.

Serum PSA has not been in routine use for female patients, levels considered to be normal are not well clarified. Age specific ranges are also not available and moreover, there is possible ethnic variability, in black and white women-0.0-0.36µg/L, in Hispanics-0.0-2.2µg/L.17 Therefore, no cut off for PSA was used to define normal in study subjects. Polycystic ovary was considered by presence of 12 or more follicles in each ovary measuring 2-9mm in diameter, and/or increased ovarian volume (>10ml).12

Serum PSA level and testosterone was analyzed by chemiluminescent immunometric assay, which was carried on the ADVIA Centaur PSA assay. USG was done by the same sonologist for each subject. All data were expressed as frequencies and mean (±SD) as applicable. Student's t-test and one way ANOVA for continuous variables like PSA. Chi-square test for discrete variables (PSA under various cut-offs) were used. Correlation among variables was assessed by using Pearson's correlation tests. Pvalues≤0.05 were considered as significant.

RESULTS:

Anthropometric characters of study subjects are shown in Table1. Age range of 50 PCOS subjects were 16-35years (mean±SD, 22.31±4.20years) and their BMI ranged from 21.60 to 37.10kg/m2 (mean±SD,27.898±4.26kg/m2). Control subjects ranged in age from 23-34years (mean±SD, 27.01±3.09years) with BMI ranging from 16.90-26.20kg/m2 (mean±SD, 22.01±2.12kg/m2). The differences of PCOS and controls in terms of age and BMI were significant (p=<0.001 for both). Waist circumference as well as both systolic and diastolic blood pressure were significantly higher in PCOS group than

those of control group (p<0.001 for all). None of the control subjects exhibited any acne, acanthosis nigricans, hirsutism or menstrual irregularities.

Table 2 depicts serum PSA and testosterone level which were significantly higher in PCOS subjects than control (0.05±0.01 vs. 0.04±0.02ng/ml; p<0.02; 40.16±20.98 vs.28.91±13.01ng/dl; p=<0.01 respectively). Holding cut-off for serum testosterone at 53ng/dl, all control subjects fell in the group having testosterone <53ng/dl whereas in the PCOS, 8(20%) subjects had testosterone level ≥53ng/ dl (p=<0.02, Table 3). As shown in Table 4, PSA level was similar in the groups of PCOS patients divided by testosterone level at cut off of 53 ng/dl (0.0521±0.006 vs. 0.0527±0.014ng/ml, p=0.910). Similar to that as grouped under testosterone cut off, PSA level in subjects with or without hirsutism in PCOS group did not differ statistically (0.0528±0.010 vs. 0.0525±0.012ng/ml, p=0.940, Table 5). As displayed in Table 6, PSA level seemed a bit lower trend with increased age group, but difference was statistically non significant (p=0.835).

Table 1: Characteristics Of Study Subjects

Variables	PCOS(n=50)	Control (n=50)	p
Age(mean±SD, year)	22.31±4.2	27.01±3.09	<0.0001
BMI(mean±SD, kg/m2)	27.898±4.26	22.01±2.12	<0.0001
Waist circumference(cm)	85.62±9.12	73.96±5.47	<0.0001
Systolic Pressure	120.82 ±13.11	100.69±7.41	<0.0001
Diastolic Pressure	75.78±9.7	68.12±6.12	<0.0001
Acne	11(27.2)	0	
Acanthosis nigricans	27(66.9)	0	
Hirsutism	32(79.9)	0	
Menstrual irregularity	36(89.8)	0	

(Data were expressed as frequency, percentage, mean±SD)

Comparison between PCOS and Control done by Student's t-test and Chi square test BMI, body mass index; PCOS, polycystic ovary syndrome

Table 2: Testosterone And PSA Levels Of PCOS And Control Subjects

Variables	PCOS(n=50)	Control(n=50)	p
S. PSA (mean±SD, ng/dl)	0.05±0.01	0.04±0.02	<0.02
S. Testosterone (mean±SD, ng/dl)	40.16±20.98	28.91±13.01	<0.01

(Data were expressed as mean±SD)
Comparison between PCOS and Control done by Student's t-test
PCOS, polycystic ovary syndrome; PSA, prostate specific antigen

Table 3: Frequency Of PCOS And Controls By Testosterone Cut-off At 53ng/dl

Group	PCOS(n=50)	Control(n=50)	p
>53ng/dl	18(20)	0	
< 53ng/dl	32(80)	50(100)	0.019
Total	50	50	

(Data were expressed as frequency, percentage)
Comparison between PCOS and Control done by Chi-square test
PCOS, Polycystic ovary syndrome

Table 4: PSA Level(ng/ml) In PCOS And Control Divided By Testosterone Cut Off At 53ng/dl

Testosterone	PCOS(n=50)	Control(n=50)
>53ng/dl	0.0521±0.006	-
< 53ng/dl	0.0527±0.014	0.0471±0.006
p	0.91	

(Data were expressed as mean±SD)
Comparison between PCOS and Control done by Student's t-test
PCOS, polycystic ovary syndrome; PSA, prostate specific antigen

Table 5: Correlations Of PSA With BMI, Age, Testosterone, Hirsutism And Polycystic Appearing Ovaries In USG In PCOS And Control

Determinants of "r"	PCOS (n=50)		Control (n=50)	
	r	p	r	p
BMI vs PSA	0.071	0.634	0.121	0.541
Age vs PSA	-0.005	0.971	-0.101	0.622
Testosterone vs PSA	-0.074	0.641	0.070	0.732

PCOS, polycystic ovary syndrome; BMI, body mass index; PSA, prostate specific antigen.

DISCUSSION:

PSA is synthesised and secreted by cells having definite hormone receptors which are under steroidal regulation.[9] This has been confirmed from our study demonstrating a significant rise in serum PSA in PCOS patients. The most pertinent finding of present study is the significant positive correlation between serum PSA and total testosterone (Table 3). This may be attributed to the hypothesis that expression of PSA gene is under androgenic regulation.[10] Similar observations have also been registered by other workers who observed a higher PSA in hyper androgenic states and after testosterone therapy.[9,10] Impact of PSA level as marker of PCOS is still far from clear. In the context of observed higher level of PSA in patients with PCOS by some investigators,10 present study was conducted to measure and compare the serum PSA level in women suffering from PCOS with that of controls. It was observed that there was statistically significant difference of PSA level between the PCOS and control subjects. This result is in agreement with the findings of some other studies.[10-13]

Both PSA and testosterone level were found significantly increased in PCOS than those of control in the present study. But, contrary to the expectation, PSA levels in PCOS women did not significantly correlate with either testosterone or hirsutism.[14] It is further accompanied by the observation that the level of PSA was statistically similar in subjects with or without hirsutism as well between the subgroups of PCOS divided by testosterone at cut off of 53ng/dl. Similar results were found by Obeizu et al and they did not find any association between hirsutism score and urinary PSA which they explained as a fact that PSA reflects the level of circulating androgen but the hirsutism score is mostly attributable to either circulating or local androgens produced and acting in the skin.[15]

Clinical manifestations like hirsutism, acne, acanthosis and menstrual irregularities are well known features of PCOS, which were also significantly higher in our PCOS group. Nearly similar frequencies were observed in other studies done in our population previously.[14,15] Present study did not evaluate the androgen and PSA level at various phases of menstrual cycle. Therefore, whether any variation is present in different phases of menstrual cycle is beyond the capacity of this study to discern. This study also did not follow the PCOS patients for change of PSA on treatment.

CONCLUSION:

We conclude that PSA level is increased in PCOS than that of control. It may be a promising marker in patients with PCOS in future. Relationship of PSA and testosterone as well as hirsutism is not yet fully clear and need wide scale study for exploration of their exact relationship in PCOS.

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Conflict Of Interest: None declared

Abbreviations: PSA, prostate specific antigen; PCOS, polycystic ovary syndrome

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