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Original Research Article

Determination of Prostatic Specific Antigen (PSA) Level in Patients with Polycystic Ovary Syndrome (PCOS)

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Abstract

Introduction: The polycystic ovary syndrome (PCOS) is a reproductive endocrine disorder, clinically characterized by oligo-ovulation/chronic anovulation, menstrual irregularities and hyperandrogenism.

Objectives: The aim of this study was determined and compare the concentration PSA in 50 patients with PCOS and 50 healthy female controls.

Result: The present observation showed that the mean level of PSA concentration in case (group I) was found to be (0.84 ± 0.30 ng/ml) and in control (group II) was found to be (0.53 ± 0.29 ng/ml). Statistical analysis showed that $p < 0.05$.

Conclusion: The PSA concentration in PCOS patient was found to be higher than that of healthy controls, and the difference was statistically significant. A comparatively elevated PSA level in PCOS women as compared to normal women is an indication of hyperandrogenism.

Key words: polycystic ovary syndrome (PCOS), prostatic specific antigen (PSA), Androgen, FSH, LH, Diabetes mellitus (DM), Ultra-sonography (USG).

Introduction

The polycystic ovary syndrome (PCOS) is a reproductive endocrine disorder, clinically characterized by oligo-ovulation/chronic anovulation, menstrual irregularities, hyperandrogenism (such as hirsutism and acne), hyperinsulinemia, insulin resistance type II Diabetes Mellitus, endometrial carcinoma and obesity [1]. PCOS is a heterogenous disorder. PCOS affected approximately 5-10% in women of reproductive age [2,3].

Androgen suppression after diagnosis of hyperandrogenism remains the primary basis for PCOS treatment in patients who do not wish to have immediate fertility [4]. An elevated number of cystic atretic follicles that are present in the periphery of the ovary and increased insulin growth factor I (IGF-I) and insulin levels that stimulate androgen synthesis in vivo and in vitro, are said to be responsible for hyperandrogenism in PCOS. Hyperinsulinism also serves to decrease circulating levels of sex hormone binding globulin (SHBG), resulting in higher levels of free androgens [5,6]. Increase levels of luteinizing hormone (LH) and successively influences on the thecal compartment of the ovary may play an additional role in the setting up of clinically apparent hyperandrogenism.

Prostate-specific antigen (PSA) was reported and find to be a 33-kDa serine protease that is primarily a product of prostatic tissue and secreted into the seminal plasma. PSA is used as a highly specific and valuable marker of prostatic adenocarcinoma regarding the screening, diagnosis and monitoring of the disease. Recent development of ultrasensitive assays demonstrated PSA in a wide variety of female tissues and fluids such as the ovary, breast, amniotic fluid, and milk, which provided worthy implications, especially in diagnosis and clinical follow-up of breast cancer cases [7,8].

In PCOS cases, serum PSA have been reported to be elevated. PSA production seems to be associated by steroid hormones such as progestin, androgens, and glucocorticoids. Zarghamiet al. [9] have showed that

PSA is up-regulated by androgens in females. PSA has been detected as a potential novel marker not only in PCOS women but also in hirsute women with hyperandrogenism [10,11].

Material and methods

This study was designed to compare PSA in PCOS patients and healthy controls. The study included determination of PSA status in 100 subjects, among them 50 were PCOS patients in hadoti region of Rajasthan, and 50 healthy controls without any clinical symptoms or disease. For diagnosis of Polycystic ovary syndrome (PCOS), history and physical findings with supportive biochemical evidence were taken as criteria and sample was collected from gynaecology department OPD SRG Hospital Jhalawar Medical College, Jhalawar (Rajasthan).

Inclusion Criteria and Exclusion Criteria in Our Study

Inclusion criteria are

- In clinical criteria, clinical hyperandrogenism and menstrual irregulaties.
- PCOS was diagnosed based on **Rotterdam criteria.**
- Clinical and/or biochemical hyperandrogenism
- Chronic oligoanovulation
- Polycystic ovaries on pelvic ultrasound examination.
- Patients with a history of hirsutism.
- Patient with USG finding of PCOS.

Exclusion criteria are

- Patient with Breast and other ovarian disease.
- Patient with recent pregnancy.
- Use of oral contraceptives, ovulatory agents, antidiabetic medications and glucocorticoids within three months prior to enrolment.

During this study, the conditions of ethics and the regulation were followed and no experiments were carried out to impair the health of patients. The study was approved by Ethical Committee of Jhalawar Medical College, Jhalawar (Rajasthan), and patients involved in the study agreed to be included in the study by signing informed written consents.

Statistical Analysis

Statistical analysis of data was done by the help of SPSS software (version 23.0). Unpaired – t test was used in data analysis for this study. P value <0.05 was consider as significant.

Results

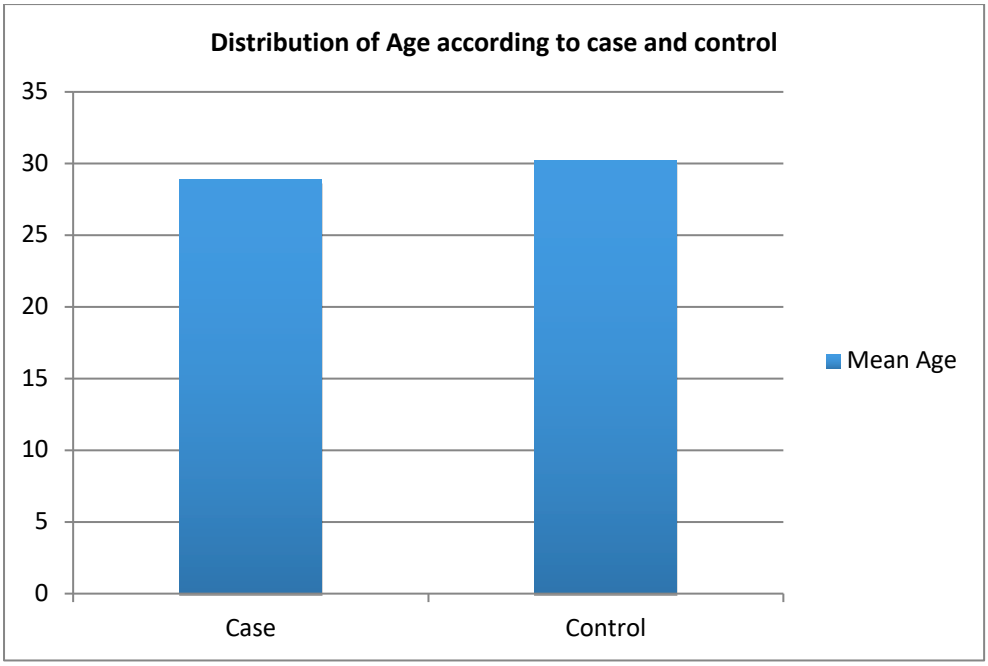
In order to □ulfil the objectives of the present study, there were 100 subjects included in our study, which were divided into two categories of groups. The first category of groups included the case group, which consisted of 50 PCOS patients, and the control group, which consisted of 50 healthy subjects. The statistical analysis of the data was done using SPSS software (version 23.0). The Chi – square test and the unpaired-t test, were used in data analysis. The data in the study were expressed as mean ± SD, and p value < 0.05 was considered statistically significant.

Distribution of Age According to Case and Control

Table 1: Comparison of age between Group – I and Group – II

Group	N	Mean	Std. Deviation	t-value	p-value
Group – I Cases	50	28.8800	6.04284	1.027	0.307
Group - II Controls	50	30.2000	6.79436		

The comparison of age between cases and controls was statistically analysed using an unpaired – t test. The mean age in PCOS patients was found to be (28.88 ± 6.04 years). The mean age in healthy controls was found to be (30.20 ± 6.79 years). Statistical analysis showed that the p – value was 0.307, i.e. (p > 0.05) therefore, the age difference in both groups was statistically insignificant.



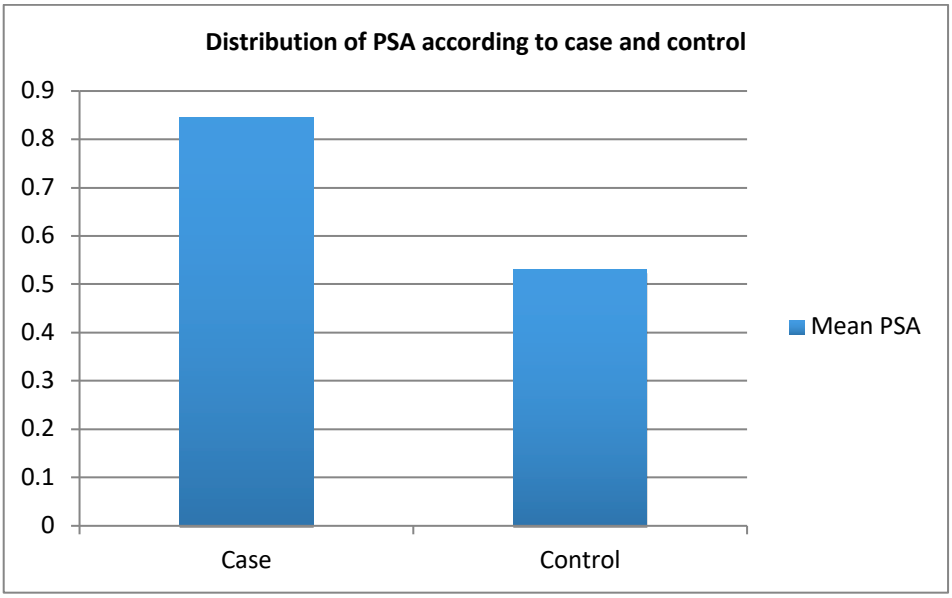
Graph – 1: Comparison of age between Group – I and Group – II

Distribution of PSA According to Case and Controls

Table 2: Comparison of PSA between Group – I and Group –II

Group	N	Mean (IU/L)	Std. Deviation	t -value	p-value
Group – I Cases	50	0.8458	0.30334	5.240	**<0.0001***
Group- II Controls	50	0.5314	0.29660		

The comparison of serum PSA concentration in cases and controls was statistically analysed using an unpaired – t test. The mean serum PSA concentration in patients was found to be (0.84 ± 0.30 ng/ml). The mean serum PSA concentration in healthy controls was found to be (0.53 ± 0.29 ng/ml). Statistical analysis showed that the p – value was **<0.0001*,** therefore the difference in serum PSA concentration in both groups was statistically significant.



Graph 2: Comparison of PSA between Group – I and Group – II

Discussion

The present study was conducted of 100 subjects, which were divided into two categories of groups. Group (I) composed of 50 Polycystic ovary syndrome (PCOS) patients (aged from 18-45 years) in the hadoti region of Rajasthan; Group (II) composed of 50 participants (aged from 18-45) who were healthy controls. The sample size for the study was 100. History and other clinically relevant data were collected from the participants.

The age distribution in both groups was statistically compared using an unpaired – t test. Concentration of Prostatic Specific Antigen (PSA) were measured in both groups using SNIBE MAGLUMI-1000 in biochemistry Laboratory, Department of Biochemistry, Jhalawar Medical College, Jhalawar (Rajasthan). The results were compared statistically using an unpaired – t test. Statistical analysis for all types of comparisons was done using SPSS software (version 23.0). The data in this study was expressed in terms of mean ± SD, and $p < 0.05$ was statistically significant.

PSA Concentration in Cases and Controls

The comparison of PSA concentration between Group – I and Group – II is presented in Table – 2 and Graph – 2. The present observation showed that the mean PSA concentration in PCOS patients was (0.84 ± 0.30 ng/ml), whereas mean serum PSA concentration in healthy controls was (0.53 ± 0.29 ng/ml). Statistical analysis showed that p – value was **<0.0001** when compared between the two groups; therefore, the difference in serum PSA concentration in both groups was statistically significant.

Thus, it was concluded that PCOS patients have higher serum PSA concentration than healthy controls, but the difference was statistically significant. The mean value of serum PSA in PCOS patients and healthy controls indicated that their concentration is within the reference interval for serum PSA (<4ng/ml).

In **2019**, researchers **Zeng-Hong Wu, Yun Tang** et al conducted a study and found that the meta-analysis suggested that PCOS was significantly linked to serum tPSA or fPSA levels (SMD = 0.81, 95% CI: 0.58 to 1.04; P < 0.01) and that in PCOS patients the serum PSA was increased when compared with controls. Furthermore, serum PSA levels are increased in hirsute patients and associated with the degree of hyperandrogenism [12-14]. However, similar result showed in our study.

In February 2019**, Arezoo Maleki-Hajiagha, Maryam Razavi** et al presented a systematic review and meta-analysis that showed a significant increase in total PSA in women with PCOS as well as hirsutism [15]. However, in our study we can conclude that PSA level are raised in PCOS patient.

Results like our study were also seen by **Ewa Rudnicka, Stanislaw Radowicki** in July 2016 in their study done in Poland. They found that the serum TPSA concentration is significantly higher in women with PCOS than in the control group. There was also positive correlation between TPSA and total testosterone level and FAI. The fPSA was below the lower detection levels among all patients [16].

In conclusion, they confirmed that women with PCOS had significantly higher serum concentration of TPSA than healthy women, and they showed that TPSA positively correlated with testosterone and FAI; this is the evidence that the production of PSA is androgen dependent and mostly of testosterone [16].

In July **2011, Farahnaz Mardanian, Nasrin Heidari** conducted a study and they found that PSA level were higher in women with PCOS and positively correlated with LH/FSH ratio and TT, FSG, DHEAS and PSA levels in PCOS. Their results also support our study; they found that the cut-off point of PSA for the diagnosis of PCOS was greater than 0.07 ng/ml based on the sensitivity of 91%, specificity of 81.2%, positive predictive value of 81% and negative predictive value of 85% [17].

In April **2007, Birol Vural** et al, studied and determined significantly higher PSA levels in the PCOS group. According to the results of their studies, PSA is demonstrated to be upregulated by stimulatory influences of steroid hormones, namely androgens, progestins and glucocorticoids [18,19].

Conclusion

our conclusion that, in the PCOS cases, serum PSA have been reported to be elevated. PSA production seems to be associated with steroid hormones such as progestins, androgens and glucocorticoids. Zarghami et al. have showed that PSA is up-regulated by androgens in females. PSA has been detected as a potential novel marker not only in PCOS women but also in hirsute women with hyperandrogenism.

The PSA concentration in PCOS patients was found to be higher than that of healthy controls, and the difference was statistically significant. However, in both groups, the PSA concentration was within the normal limit of reference interval for PSA (<4ng/ml).

References

1. GuzickD. Polycystic ovary syndrome: sympto-matology, pathophysiology, and epidemiology. Am J Obstet Gynecol. 1998;179:89–93.
2. Knochenhauer ES, Key TJ, Kahsar-Miller M, et al. Prevalence of the polycystic ovary syndrome in unselected black and white women of the

southeastern United States: a prospective study. J Clin Endocrinol Metab. 1998;83(9): 3078–82.
3. Glintborg D, Andersen M. An update on the pathogenesis, inflammation, and metabolism in hirsutism and polycystic ovary syndrome. Gynecol Endocrinol. 2010;26(4):281–96.
4. Vural B, Ozkan S, Bodur H. Is prostate-specific antigen a potential new marker of androgen excess in polycystic ovary syndrome? J ObstetGynaecol Res. 2010;33(2): 166–73.
5. Azziz R. Androgen excess is the key element in polycystic ovary syndrome. FertilSteril 2003 ; 80: 252–254.
6. Lobo RA. What are the key features of importance in polycystic ovary syndrome? FertilSteril 2003; 80: 259-261.
7. Escobar-Morale HF, Serrano-Gotarredona J, Avila S, VillarPalasi J, Varela C, Sancho J. The increased circulating prostatespecific antigen concentrations in women with hirsutism do not respond to acute changes in adrenal or ovarian function. J Clin Endocrinol Metab 1998; 83: 2580–2584.
8. Burelli A, Cionini R, Rinaldi E et al. Serum PSA levels are not affected by the menstrual cycle or the menopause but are increased in subjects with polycystic ovary syndrome. J Endocrinol Invest 2006; 29: 308–312.
9. Zarghami N, Grass L, Diamandis EP. Steroid hormone regulation of prostatespecific antigen gene expression in breast cancer. Br J Cancer. 1997; 75:579–88.
10. Gullu S, Emral R, Asik M, et al. Diagnostic value of prostatic specific antigen in hirsute women. J Endocrinol Investig. 2003; 26:1198.
11. Negri C, Tosi F, Dorizzi R, et al. Antiandrogen drugs lower serum PSA levels in hirsute subjects: evidence that serum PSA is a marker of androgen action in women. J Clin Endocrinol Metab. 2000;85:81–4.
12. Cleutjens KB, van Eekelen CC, van der Korput HA, Brinkmann AO, Trapman J. Two androgen response regions cooperate in steroid hormone regulated activity of the prostate-specific antigen promoter. J Biol Chem. 1996;271: 6379–88.
13. Yu H, Diamandis EP, Monne M, Croce CM. Oral contraceptive-induced expression of prostate-specific antigen in the female breast. J Biol Chem. 1995;270:6615–8.
14. Melegos DN, Yu H, Ashok M, Wang C, Stanczyk F, Diamantidis EP. Prostatespecific antigen in female serum, a potential new marker of androgen excess. J Clin Endocrinol Metab. 1997;82:777–80.
15. ArezooMaleki-Hajiagha et al; Serum Prostate-Specific Antigen Level in Women With Polycystic Ovary Syndrome: A Systematic Review and Meta-analysis; HormMetab Res 2019; 51: 230–242
16. EwaRudnicka, Stanislaw Radowicki et al. Prostate specific antigen (PSA) in diagnosis of polycystic ovarian syndrome. Gynecological Endocrinology. 2016-07-19; 1473-0766; DOI: 10.1080/ 09513590. 2016.1200552
17. FarahnazMardanian, Nasrin Heidari. Diagnostic value of prostate-specific antigen in women with polycystic ovary syndrome. J Res Med Sci. 2011-07-24; ; 16(8): 999-1005
18. Katsaros D, Melegos DN, Diamandis EP. Prostate-specific antigen production by breast tumors after induction with oral contraceptives. Clin Biochem 1998; 31: 285–288.
19. Goh VHH. Breast tissues in transexual women-a nonprostatic source of androgen upregulated production of prostatespecific androgen. J Clin Endocrinol Metab 1999; 84: 3313– 3315.