



COMPARATIVE STUDY OF SERUM ANDROGEN LEVEL BETWEEN NORMAL FEMALE & FEMALE WITH POLYCYSTIC OVARIAN SYNDROME (PCOS).

Physiology

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ABSTRACT

Circulating testosterone level is high in PCOS. The source of testosterone is either ovary for adrenal or both. In ovary androgen production is controlled by LH. Where LH is excessive and FSH is suppressed, this results in an imbalance leading to increased androgen production with normal or suppresses oestrogen production; particularly oestradiol. Extensive evidence confirms that adrenal androgen excess is also common in PCOS. The aim of the present study is to estimate the serum androgen level in patients of PCOS & control group. Significant elevation of plasma Androgen level is observed in PCOS cases then normal control group. This will help to understand the pathophysiology of polycystic ovarian disease.

KEYWORDS

PCOS - polycystic ovarian syndrome, LH - Luteinizing hormone, FSH - follicle stimulating hormone

INTRODUCTION:

Polycystic ovarian syndrome (PCOS) also known as Stein Leventhal syndrome is a condition that affects many women during their childbearing age. It is the most prevalent endocrinopathy in women and the most common cause of menstrual disturbance during the reproductive age. It is also one of the leading cause of female infertility. Symptoms of PCOS are related to anovulation and androgen excess. Classic symptoms include menstrual irregularity, may be associated with infertility, hirsutism, acne and obesity. Pathophysiologically PCOS is characterized by disruption of the regular processes of ovulation and associated with hyperandrogenemia, normal or elevated oestrogen level, raise LH secretion with alteration of the normal relationship between LH and FSH, leading to raise LH : FSH ratio. On histological examination the ovaries show atretic follicles, theca cell hyperplasia, & generalized increased in stroma. On USG examination the ovaries are characterized by peripheral distribution of multiple sub capsular cysts.

Estrogen level is normal or elevated. This estrogens arises from the ovary and from the conversion of androstenedione to oestrone mainly by the fatty tissue. It is the chronically high level of oestrone, together with a cyclical nature of oestradiol secretion by the ovary and the elevated level of the unbound oestradiol provide the abnormal feedback to the pituitary and hypothalamus, which lead to Excess LH secretion and suppress FSH secretion in response to GnRH release.

Circulating testosterone level is high in PCOS. The source of testosterone is either ovary for adrenal or both. In ovary androgen production is controlled by LH. Where LH is excessive and FSH is suppressed, this results in an imbalance leading to increased androgen production with normal or suppresses oestrogen production; particularly oestradiol. Extensive evidence confirms that adrenal androgen excess is also common in PCOS.

PCOS may represent a common end point for any mechanism bringing about ovulation arrest in the presence of oestrogen level which are not suppressed. In addition to an intrinsic ovarian abnormality other potential initiating abnormalities include, excess adrenal androgen secretion, obesity, hyperinsulinemia usually associated with insulin resistance.

MATERIAL & METHOD:

The study has been done of 50 cases of PCOS & 30 cases of control during January 2007 to December 2007, in Sheth Chinai Maternity Hospital, Ahmadabad. Clinical details of patients were recorded in history taking, examination of patient for any hormonal imbalance. Patients were investigated for hormonal assay during follicular phase of cycle. USG examination is also done for ovulation study. Thus by thoroughly examining & investigating the cases, we could diagnosed 50 cases of PCOS & the data were compared with 30 normal cases.

Observation & Result:

SERUM TESTOSTERONE LEVEL

S. Testosterone (ng/ml) level	PCOS CASE		CONTROL GROUP	
	No. of cases	Percentage	No. of cases	Percentage
Upto 0.7	16	32	27	90
More than 0.7	34	68	9	10
Mean S. Testosterone (ng/ml) level	0.81	0.44		

In our present study, serum Testosterone level is more than normal value in 68 % of patient. Also the mean serum Testosterone value in PCOS group is higher than control group. These finding confirm that circulating androgenic steroids are higher in PCOS. Significant elevation of virtually all androgenic steroids have been found in women with PCOS in a study of (Bardin et.al, 1988 Lobo et.al 1989). The percentage of elevated level of serum Testosterone varies with different studies from 69 % (De Vane et.al, 1975) to 75 % (Mc Kenna et.al, 1996).

DISCUSSION:

The primary abnormality in PCOS could reside within the ovaries or the ovarian abnormalities could be secondary to extra-ovarian disturbances (Mc Kenna, 1988 Frank, 1989, Poretshy & Piper 1994). Extensive evidence confirms that adrenal androgen excess is common in PCOS. This has been shown by means of calculated adrenal androgen production rate and the demonstration of the excessive androstenedione, DHEA and testosterone response to stimulation by exogenous ACTH or following stimulation of the endogenous ACTH secretion using Metirapone (Mc Kenna, 1988). The mechanism underlying the access adrenal androgen production has not been established. (Mc Kenna and Cunningham, 1991). The possible mechanism include excess stimulation of adrenal gland by factors involved specifically in the control of androgen production.

Alternatively, there is evidence that the metabolic clearance of cortisol is increase in PCOS. Two different sets of enzymes have been demonstrated to be excessively active in this condition, both of which are involved in the metabolism of cortisol i.e. 5 alpha reductase & 11 beta hydroxysteroid dehydrogenase (Stewart et.al 1990, Rodin et. al, 1994). As a consequence of increase cortisol metabolism, plasma cortisol level will tend to fall and as a compensatory response ACTH secretion increases. This will correct falling cortisol but will bring about a rise in androgen level as ACTH undoubtedly play an important, at least facilitatory, role in adrenal androgen production and secretion.

An additional school of thought believes that the adrenal and ovary share a defect in the activity of the 17- alpha hydroxylase cytochrome P450 enzyme system. When this system is induce, there may be excess production of 17- hydroxyprogesterone and excess conversion of 17 - hydroxyprogesterone to androgen. As 17-20 lyase activity is also controlled by the same enzyme. (Barnes & Rosenfield, 1989). Therefore, several mechanisms exist whereby adrenal androgen production may be enhanced in PCOS and supply substrate for

peripheral conversion of androgen to oestrone. Oestrone may then perturb gonadotrophin secretion leading to changes of PCOS. Alternatively, adrenal androgen excess may have a direct effect on the ovary, disrupting the normal Graafian follicle development.

CONCLUSION:

The present study identifies the association of raised plasma Androgen levels with PCOS. Circulating testosterone level is high in PCOS. The source of testosterone is either ovary for adrenal or both. In ovary androgen production is controlled by LH. Where LH is excessive and FSH is suppressed, this results in an imbalance, leading to increased androgen production. Extensive evidence confirms that adrenal androgen excess is also common in PCOS.

REFERENCES:

1. Adams J, Polson DW & Frank S (1985). Prevalance of PCOS in anovulatory women. *Journal of Endocrinology*.
2. Balen AH; Conway GS et al, PCOS: The spectrum of the disorder in 1741 patients "Human reproduction Journal August 1995 England."
3. Barnes RB, Pathophysiology of Ovarian Steroid secretion in PCOS, *Seminars in reproductive endocrinology* (U. S.) May, 1997.
4. Best & Taylor's Physiological basis of Medical Practice, Twelfth Edition.
5. Chang RJ, Katz SE. Diagnosis of PCOS *Endocrinology and Metabolism clinics of North America*, June 1999.
6. Chaudhary: *Concise Medical Physiology* Edition third.
7. Clayton RN et. al, 2000 Sex Steroids, adiposity and smoking in the pathogenesis of idiopathic hirsutism and PCOS.
8. Cunningham SK, Mc Kenna TJ 1994, Dissociation of adrenal androgen and cortisol secretion in Cushing's syndrome.
9. Dewailly D, Definition and significance of PCOS. *Journal Articles* June 2007.
10. Edman CD and Mac Donald (2008). Effect of obesity on conversion of plasma androstenedione to oestrone in ovulatory and anovulatory young women, *American Journal of Ob & Gyn*.
11. Erricson GF, et. al, Functional studies of aromatase activity in human granulosa cells from normal and polycystic ovaries. *Journal of Clinical Endocrinology and Metabolism* (2001).
12. Flad TM et al. 1993: Gonadotropin, androgen and estrogen abnormalities in PCOS occurs secondary to anovulation and progesterone deficiency *Journal Endocrinol* 139:32.
13. Ganong WF, *Review of Medical Physiology*. 14th Edition.
14. Gonzalex F: Adrenal involvement in PCOS seminars in reproductive endocrinology (U.S.) May 1997, *Deptt. Of Gyn & Obst*.
15. Goudas VT, Dumesic DA. Polycystic Ovary Syndrome *Endocrinology and Metabolism clinics of North America*. December 1997.
16. Guyton and Hall; *Textbook of Medical Physiology*. Edition Eleventh.
17. Kahn JA, Gordon CM. Polycystic Ovary Syndrome *Journal article*. *Adolescent Medicine*, (U.S.) June 1999.
18. Leondirs MP, Benga SL. Role of GnRH drive in the pathophysiology of PCOS. *Journal Article*. *Journal of Endocrinology investigation*, July, August, 1998.
19. Marantides D, Management of PCOS. *Journal Article in Nurse Fractioned*. December 1997.
20. Mc Kenna TJ, Hayer FJ. Recent advances in the diagnosis and treatment of PCOS. *Article published in Recent advances in Ob & Gyn*. 1997-19.
21. Ovesen PG et. Al, PCOS clinical presentation and treatment. *Journal Article* (2006).
22. San Diego, La Jolla. Inappropriate gonadotropinn secretion in PCOS. *Journal of clinical endocrinology and metabolism*, November 1997.
23. Stephen Franks et. al, Ovulatory disorders in women with PCOS. *Article in clinics in Obst. & Gynaec*. September 1985.