DETERMINATION OF SERUM GONADOTROPIN AND TESTOSTERONE LEVELS IN MALE INFERTILITY

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ABSTRACT

Objective: *To determine the levels and ratios of serum LH, FSH and Testosterone, among men having history of infertility.*

Material and Methods: *A non-interventional descriptive study was carried out in the Department of Public Health Laboratories Division, NIH, Islamabad from January 2004 to December 2005. Two hundred fifty infertile men, classified as azoospermic (50), oligozoospermic (75), asthenozoospermic (50) and normozoospermic (75) were studied for serum LH, FSH and Testosterone, in addition to 50 proven fathers.*

Results: *The respective FSH (mIU/ml), LH (mIU/ml) and Testosterone (nmol/L) levels for the groups were 22.924.15, 13.852.33 and 11.860.70 (Azoospermia), 16.823.79, 10.921.22 and 11.88±1.06 (Oligozoospermia), 3.220.61, 3.921.17 and 16.24±2.05 (Asthenozoospermia) while in normozoospermic men were 5.530.52, 7.400.60 and 17.29+1.02, The proven fathers group had 7.74±0.71 mIU/ml LH, 6.75+1.06 mIU/ml FSH and 15.88±1.15 nmol/ml testosterone, respectively.*

The LH/FSH and FSH/LH ratios were 0.77±0.08 and 1.84±0.22 (Azoospermia), 1.78±0.41 and 1.47±0.18 (Oligozoospermia), 1.51±0.46 and 1.28±0.28 (Asthenozoospermia) while in normozoospermic and proven fathers were 1.55±0.16 and 0.85±0.10,1.67±0.22 and0.94±0.12. Similarly, the LH/T and T/LH ratios were 1.17±0.28 and 0.86±2.70 (Azoospermia), 0.92±0.28 and 1.08±0.17 (Oligozoospermia), 0.30±0.10 and 4.14±10.4 (Asthenozoospermia) and in normozoospermic were 0.42±0.08 and 2.34±0.48.The ratios observed for T/FSH and FSH/T were 0.52+0.17 and 1.93+5.95 (Azoospermia), 0.71+0.28 and 1.42+3.57 (Oligozoospermia), 5.04±3.38 and 0.20±0.30 (Asthenozoospermia) and in normozoospermic men were 2.82±0.90 and 0.35±1.12, while in proven fathers were 4.09±0.57and 1.04±0.53 respectively.

Conclusion: *Altered/disturbed concentrations of gonadotropins and androgenic hormones and their ratios cause infertility, since all these hormones act synergistically.*

Key words: *Testosterone, FSH, LH; infertility; Male Factor; Pakistan.*

INTRODUCTION

Infertility is a common disorder and nearly one out of every 6-8 couples suffers from it at any given time. Infertility among couples in their respective age is more common than hypertension, diabetes, heart diseases and even the common flu.[1] Globally, approximately 10-15% couples seek medical help for the problem of infertility. In 20-25% cases the problems are attributable to the male partner, while, 30-40% represent female factor. Approximately 30% of cases are associated with both partners and in 15% no specific factor can be identified.[2] In Pakistan where potency is considered a proof of normal fertility, it is usually the wife who bears the blame and is often maltreated for the offence of not bearing children. The husband, who should also be investigated simultaneously either never submits himself for evaluation or is investigated only at the end.[3] In general infertility is defined as the failure to conceive after 1 year of regular unprotective intercourse with the same partner. However, the term “Infertility” implies a definitive inability to conceive. Therefore, couples who do not conceive in >1 year should be regarded as sub fertile. According to these definitions, approximately 14%

MEAN (±SEM) SERUM LH, FSH AND TESTOSTERONE LEVELS

Group	LH (mIU/ml)	FSH (mIU/ml)	Testo (nmol/L)
Azoospermia	13.85 ± 2.33[a]	22.92 ± 4.15[c]	11.86 ± 0.70[b]
Oligozoospermia	10.92 ± 1.22[d]	16.82 ± 3.79[a]	11.88 ± 1.06[a]
Asthenozoospermia	3.92 ± 1.17[b]	3.22 ± 0.61[b]	16.24 ± 2.05[d]
Normozoospermia	7.24 ± 1.02[d]	5.53 ± 0.52[d]	17.29 ± 1.02[d]
Proven fathers (control)	7.44 ± 0.71	6.75 ± 1.06	15.88 ± 1.15

Table 1

d = Non-significant($p>0.05$)
a = Significant($p<0.05$)
b = Significant($p<0.01$)
c = Significant($p<0.001$)
Normal levels of Testosterone = 5.2-22.9 nmol/L
Normal level of LH = 2-12 mIU/ml.
Normal levels of FSH = 1-12 mIU/ml.

of the couples are sub fertile.[4] To be more exact the term subfertile means a male who failed to conceive after I year of regular unprotected intercourse with the same partner and who had a sperm count of less than 20 million/ml.[5] Various hormones have their role which affect the spermatogenesis. Testosterone secreted by leydig cells in testis, is essential for growth and division of germinal cells in forming spermatozoa. Luteinizing hormone (LH), secreted by anterior pituitary gland, stimulates the leydig cells to secret testosterone. Follicle stimulating hormone (FSH) secreted by anterior pituitary gland, stimulate sertoli cells, without this stimulation conversion of spermatids to spermatozoa will not occur.[6] The successful and complete male germ cell development is dependent on the balanced endocrine interplay of hypothalamus, pituitary and the testis. Gonadotrophin releasing hormone (GnRH) secreted by the hypothalamus elicits the release of gonadotrophins i.e. follicle stimulating hormone (FSH) and lutenizing hormone (LH) from the pituitary gland.[7] FSH binds with receptors in the Sertoli cells and stimulates spermatogenesis, while LH stimulates the production of testosterone in Leydig cells, which in turn may act on the Sertoli and peritubular cells of the seminiferous tubules and stimulates spermatogenesis.[8] The failure of pituitary to secret FSH and LH will result in disruption of testicular function leading to infertility. Testosterone, estradiol and inhibin control the secretion of gonadotrophins through feed back mechanism.[9] Status of the male infertility can be assessed through simple semen examination, followed by hormonal profile if required.[6] Almost 40% of the total male infertility is mainly because of azoospermia.[10] Absence of spermatozoa in the semen ejaculate is called "azoospermia", count less than 20 million/ml "Oligospermia" and density of 20 million/ml but motility of less than 50% is called "asthenospermia".[11] Male infertility is associated with a reduction in the quantity of functional sperms.[12] Decrease in sperm density, eventually leading to azoospermia, has been found to be associated with raised FSH, LH and normal or low testosterone level.[13] Hormonal disturbances result in almost 1/6 married couples not having any progeny, and the male partner being responsible in nearly half of such cases.[14] Determination of concentration of LH and Testosterone is essential for the evaluation of pituitary gonadal axis and in the evaluation of infertility.[15] Studies on determination of ratios of various hormones are rare.[16]

The present study, although preliminary in nature, aims to determine the levels and ratios of serum LH, FSH and Testosterone, among men having history of infertility and to see the flipped condition in various groups of sub fertility, after comparing with proven fathers.

MATERIAL AND METHODS

A total of 250 selected male subjects with a history of infertility for the duration of more than 1 year were included. These patients had been referred by gynecologists, urologists, and general practitioners from different parts of the country, for semen and hormone analysis. The study was carried out at Reproductive Physiology /Health, Public Health Laboratory Division, National Institute of Health Islamabad. A total of fifty (50) cases of normal subjects belonging to the same socioeconomic status were selected as a control group. In this group the last pregnancy was recorded 1-3 years back. Clinical examinations of all the subjects were carried out and information regarding age, duration of marriage, history of infertility in the family and parity was recorded. The age of the patients was ranging from 20-55 years, however, the maximum number of patients

RATIO OF VARIOUS HORMONES INVOLVED IN SPERMATOGENESIS

Group	Ratio of LH/FSH	Ratio of FSH/LH	Ratio of LH/Testo	Ratio of Testo/LH	Ratio of FSH/Testo	Ratio of Testo/FSH
Azoospermia	0.77 ± 0.08 [c]	1.84 + 0.22 [c]	1.17 + 0.28 [d]	0.86 + 2.70 [d]	1.93 + 5.95 [d]	0.52 + 0.17 [c]
Oligozoospermia	1.78 ± 0.41 [d]	1.47 + 0.18 [a]	0.92 + 0.28 [d]	1.08 + 0.17 [b]	1.42 + 3.57 [d]	0.71 + 0.28 [c]
Asthenozoospermia	1.51 ± 0.46 [d]	1.28 + 0.28 [d]	0.24 + 0.10 [d]	4.14 + 10.4 [d]	0.20 + 0.30 [d]	5.04 + 3.38 [d]
Normozoospermia	1.55 ± 0.16 [d]	0.85 + 0.10 [d]	0.42 + 0.08 [d]	2.39 + 0.48 [d]	0.35 + 1.12 [d]	2.82 + 0.90 [d]
Proven fathers	1.67 ± 0.22	0.94 + 0.12	0.49 + 0.28	2.05 + 0.33	1.042 + 0.53	4.09 + 0.57

Table 2

d = Non-significant (p>0.05)
a= Significant (p<0.05)
b = Significant (p<0.01)
c= Significant (p<0.001)

were in the age of 25-40 years. The duration of infertility was between 1-20 years.

A detailed history was taken, in order to determine any correlation of the problem of infertility faced by couples and the status of the male partner. The patients were enquired about their abstinence period and were informed that the ideal period is 2 to 7 days. Semen samples were obtained through masturbation and were ejaculated into clean wide mouthed plastic containers, which had already been confirmed to be non-toxic to spermatozoa.

Inclusion criteria were male patients with primary and secondary infertility without treatment and having no apparent cause of male infertility were classified in different groups, based upon their semen picture. Exclusion criteria were the subjects, who had undergone pelvic surgery or hernia repair, thyroid disease patients and subjects who were on drugs e.g. antipsychotic, antihypertensive, neuroleptic etc.

The semen of the Subjects was analyzed according to WHO recommended procedure. [17] The results were categorized as normozoospermic, azoospermic, oligozoospermic and asthenozoospermic. A 5-10ml fresh blood sample was collected from anterior cubital vein from each subject under aseptic condition. The blood was transferred to a clean plain labeled tube, allowed to clot, and then centrifuged at 6000 rpm for 5 minutes in Hittech centrifuge at room temperature. The clear serum was separated and kept at 20^0C till assay. FSH, LH and Testosterone assessment was carried out using electrochemiluminescence technology by fully automatic and state of the art equipment Elecsys 2010 analyzer.[18] Samples representing the normal and pathological levels of the analytes were used for quality control. Results ± 2SD of the target value were considered acceptable. Only the batches with all controls within permissible range were accepted. Data was analyzed statistically, by application of Student "t' test, as described by Steel and Torrie.[19]

RESULTS

The results of analysis of the hormones and their respective ratios among all sub-groups are presented in Tables 1 and 2**.** In azoospermic group, both LH and FSH exhibited significant increase, with the increase in FSH being more profound (p<0.001). Testosterone levels showed a significant (p<0.01) decrease. The ratios of LH/T, FSH/LH, and FSH/T, showed an increase when compared with other groups.

In oligozoospermic group both LH and FSH were significantly high, as seen in azospermic group, with the increase in FSH being more profound (p<0.001), while testosterone levels showed a significant (p<0.05) decrease, however the increase in FSH/LH, and decrease in testosterone, was not more profound, as observed in cases of azoospermia. Due to increased LH and decreased Testosterone levels, oligozoospermic subjects exhibited significantly (p<0.05) lower ratios of T/LH. The ratios of LH/T, FSH/LH and FSH/ T were increased when compared with proven fathers group.

In cases of asthenozoospermia, both LH and FSH exhibited a significant decrease, (p<0.05), while testosterone levels decreased non-significantly (p> 0.05). Due to decreased levels of LH and FSH, asthenozoospermic subjects exhibited non-significant (p>0.05) variation in ratios of T/LH, LH/T, FSH/LH and FSH/ T, when compared with proven fathers group.

Among normozoospermic cases, the LH, FSH and testosterone levels exhibited non-significant differences, (p>0.05). Normozoospermic subjects exhibited non-significant (p>0.05) variations in ratios of T/LH, LH/T, FSH/LH and FSH/ T, when compared with proven fathers group.

DISCUSSION

FSH, LH and testosterone are prime regulators of germ cell development. The quantitative production of spermatozoa generally requires the presence of FSH, LH and

Testosterone. FSH acts directly on the seminiferous tubules, whereas lutenizing hormone stimulates spermatogenesis indirectly via testosterone.[20] From this study it is apparent, that azoospermic and oligozoospermic subjects are accompanied by a significant rise in FSH levels, and is associated with male sub-fertility. The measurement of FSH in serum may, therefore, be used with advantage in the diagnosis of spermatogenic dysfunction and also to differentiate between secretory and excretory azoospermia.[21-22] Not only the high levels of FSH and LH were observed, but low level of FSH and LH were also recorded in our study. It has been found that low levels of FSH are also associated with male infertility, as FSH is necessary for sperm maturation.[23] Low levels of LH, and hence subsequently testosterone, are also found in patients with normal spermatic maturation arrest.[24] De Krester et al[25] reported elevated levels of serum FSH, and LH with increasing severity of seminiferous epithelial damage. In the present study, elevated levels of LH were observed in azoospermic and oligozoospermic males when compared with the levels in proven fathers, as well as normospermic men.

Our results are in accordance with earlier studies[26-28], which showed gonadotrphic elevation in infertile males. In various studies elevated levels of LH in oligozoospermic and azoospermic males, when compared to normal fertile men, were also documented[29.30] which matches our findings. In the present study, the mean serum testosterone levels in both fertile and infertile men were within permissible levels. Similar observation has been recorded in earlier studies[31-33], who reported normal level of testosterone in infertile men with Sertoli cell syndrome when compared with control group, but in contrast to another study, where decreased level of testosterone was observed in infertile males[34], as sometimes there is a loss of germinal epithelium, but Leydig cells of testes remain intact. This condition is usually reflected by normal plasma testosterone level. In the present study, serum concentration of Testosterone was although within the limit, but was on lower side. In a number of related studies, It has been demonstrated that low levels of serum LH and testosterone are found in men with oligoasthenozoospermia.[35] It has been reported, that decreased secretions of LH and Testosterone in oligozoospermic men are due to prolonged half life of LH, reduced bio-active LH secretary burst amplitude, lower immunoactive ratio for LH burst amplitude, reduced bioactive /immunoactive ratio in the mass of LH secreted per burst and decreased coordinated release of bioactive LH and testosterone.[36] The suppression of testosterone secretion in infertile men could be due to deficiency of hypothalamic Gn RH, resulting in the impairment of Gonadotropin secretion from pituitary. The deficiency of LH and FSH prevents the gonads from either producing sperms or sufficient quality of testosterone.[37]

In our investigations we found a small group of subjects in infertile patients with high testosterone concentrations. It has been reported that testicular tumors prior to puberty produced high concentrations of testosterone leading to precocious puberty.[37] The significant rise in serum LH level in oligozoospermic and azoospermic males in the study, are in contrast with the findings of Saeed et al,[38] whereas, Kuku et al[39] observed elevated LH levels in 26.5% subjects and low level in 5% of infertile males, which is an agreement to our study.

In the study under discussion, different combinations were observed with respect to LH, FSH and Testosterone levels, as FSH and LH levels were high in case of azoospermia and oligozoospermia, while Testosterone levels showed significant decrease in these groups, and the variation in these levels affected the ratios.[21] It can be seen that in case of azoospermia and oligozoospermia, testosterone exhibited a significant decrease, while the increase in case of asthenozoospermia as well as normozoospermia was not significant. Significant differences in LH levels were also observed in case of azoospermia ($p<0.05$) and asthenozoospermia ($p<0.01$), being elevated in cases of azoospermia and decreased in cases of asthenozoospermia. Similarly it can be seen that in the case of azoospermia, both LH and FSH exhibited significant increases, with the increase in FSH being more profound ($p<0.001$). Significant ($p<0.01$), differences in FSH levels were also observed in case of oligozoospermia and asthenozoospermia. The LH/T ratios were observed as non-significant ($p>0.05$) in all groups in comparisons to proven fathers. Due to increased LH and decreased testosterone levels, significantly ($p<0.01$) lower ratios of T/LH were observed in cases of oligozoospermia. The FSH/LH ratio was (1.84 ± 0.22) in case of azoospermia and decreased to 0.85 ± 0.10 in case of normozoospermia. Even in conditions of oligozoospermia and asthenozoospermia, the FSH/LH ratios were well above 1.00. Due to increase in FSH levels, the FSH/LH ratios exhibited significant increases in azoospermic ($p<0.001$) and oligozoospermic ($p<0.01$) subjects, which is an accordance with our previous study.[40]

The overall results clearly indicate a significant increase in gonadotropins (FSH and LH) in azoospermia and oligozoospermia. Although there was significant decrease in the

testosterone levels in infertile males when compared with the fertile controls, the increase in the levels of gonadotrophin might have disrupted the spermatogenic process leading to the decline in the sperm count and infertility. The overall results clearly indicate significant increase of LH, in case of depleted sperm concentrations, and significant decrease in cases of asthenozoospermia. Although there was a significant decrease in the testosterone levels in azoospermic and oligozoospermic males when compared with the fertile controls, the increase in the levels of LH might have disrupted the spermatogenic process leading to the decline in the sperm count and infertility.

CONCLUSION

The study suggests that estimation of serum levels of LH and FSH and their ratios, specially the FSH/LH ratio, could be used as tool to investigate the nature of neuro-endocrine cause of infertility, as both these hormones act together. The estimation of serum levels of LH and Testosterone and their ratios, specially the LH/T ratio, could be used as tool to investigate the nature of neuro-endocrine cause of infertility, as both these hormones act in association.

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