REPRODUCTIVE HORMONES IN INFERTILITY AND INFECTION IN SUB- SAHARAN AFRICAN MALES

*CHARLES-DAVIES M. A, **OSOTIMEHIN B. O.
*Immunoassay Laborator Ebute-Metta, Lagos.,
**Department Of Chemical Pathology, University College Hospital Ibadan,

SUMMARY

The male factor is now recognised as a major contributor to a couples' infertility. The role of reproductive hormones in male infertility is however controversial. This study therefore, was designed to investigate the role of reproductive hormones in male infertility in sub-Saharan Africa.

3 groups of subjects between 18-56 years were investigated. 25, 42 and 45 were recruited in fertile, infertile and sexually transmitted diseases (STDs). Follicle stimulating hormone (FSH), luteinising hormone(LH) and prolactin were estimated using the immunoradiometric assay technique while testosterone was estimated using radioimmunoassay technique utilizing commercially available kits (ICN Biomedical Inc.).Statistical analysis was done using students t-test and anova (one way) for comparison of means.

FSH, LH, prolactin and testosterone were similar between fertile and infertile groups and between normospermic and dyspermic infertile groups. However all except, prolactin were similar in STDs and fertile groups.

Hormonal abnormalities may not be a major contributor to male infertility. However, role of prolactin in infertility and infection requires further studies.

Key words: Hormones, male infertility ,sexually transmitted diseases.

INTRODUCTION

Male factor has been implicated in about 40% of clinical infertility (1,' 2, 3). In majority of the cases, the male factor in a couples infertility can be assessed from analysis of semen (4).

The quantitative production of spermatozoa generally requires the presence of both LH/testosterone and FSH (5). Although endocrine causes are reported uncommon in both developed and developing countries (4,5) Kuku (7) in his review observed that identifiable endocrine abnormalities have be reported in 10% of infertile men in developed countries where hormonal evaluations of infertile males are routinely undertaken.

The aetiological role of endocrine infertility is hardly understood and data regarding the frequency of these abnormalities are lacking in African countries. This study therefore was designed to reevaluate the roles of reproductive hormone in infertility and sexually transmitted infection in sub-Saharan African males.

MATERIALS AND METHODS

Subjects

III subjects between 18 and 56 years were investigated. 25 of them were fertile with satisfactory semen profile and acted as controls, 44 were infertile for at least one year while 42 had incontrovertible evidence of STDs (8,9). Out of the 44 infertile men, 12 of them were normospermic while 32 were dyspermic.

*Correspondence: **M. A Charles-Davies**

Blood collection

Venous blood was collected in lithium heparin tubes, centrifuged for 5 mins at 3000rpm. Plasma was collected and stored at -20^0C for analysis.

Endocrinological analysis

FSH, LH, prolactin were estimated using immunoradiometric assay method while testosterone was estimated using radioimmunoassay technique (ICN Biomedical Inc).

Statistical analysis

Statistical analysis was carried out by means of computer statistical software-epi info 6.02. Students t –test and analysis of variance (anova)- one way were used for comparisons of means.

RESULTS

The mean concentrations of FSH, LH, prolactin and testosterone in fertile, infertile and STDs groups are shown on Table 1. None of the hormones except prolactin showed any significant difference in the comparison of means between groups ($p>0.05$).

There was significantly lower difference in prolactin in men with STDs compared with fertile and infertile men ($p=0.69$). Comparisons of hormonal levels (FSH, LH, prolactin and testosterone) between infertile men with normospermia and dyspermia did

not reveal any significant differences in all the hormones tested (p>0.05) Table 2.

Table1: Statistical comparison of Follicle stimulating hormone(FSH), Luteinizing hormone(LH), Prolactin(Prl) and Testosterone(T) between fertile, infertile and STDs subjects (using ANOVA)

	Groups				
	Fertile	Infertile	STDs	F	p
FSH (mIU/ml	N=25 18.0±2.3	n=44 19.9±2.9	n=42 12.1±2.9	2.190	0.11
LH (MiU/m	N=25 6.6±0.7	n=44 8.6±0.8	n=42 7.2 ±0.7	1.358	0.26
Prl (ng/ml)	N=24 20.1±2.1	n=42 21.4±2.1	n=42 15.0±1.2	3.851	0.02+
T(ng/ml)	N=25 7.3±0.3	n=39 7.0±0.3	n=30 7.2±0.3	0.328	0.73

values are in mean ± standard error, + = significant; n = number of subjects; p = probability

Table2: Statistical comparison of Follicle stimulating hormone (FSH), Luteinising hormone(LH), Prolactin (Prl) and Testosterone(T) between normospermic and dyspermic infertile males (using students's t-test).

	Infertile males			
	Normospermic	*Dyspermic*	T	P
FSH (mIU/ml)	n=12 14.7±3.7	n=12 21.9±3.9	1.07	0.29
LH (mIU/ml)	n=12 6.7±0.9	n=12 9.4±1.0	1.561	0.12
Prl (ng/ml)	n=12 22.3+3.0	n=12 21.1±2.7	0.267	0.79
T (ng/ml)	n=10 7.3±0.4	n=12 6.9±0.4	0.619	0.55

values are in mean ± standard error, n = number of subjects; p =probability.

DISCUSSION

Kuku (7) indicated that hormonal disorders may be responsible for some cases of oligospermia and azoospermia found in male infertile marriages. He suggested that these abnormalities are not causally related to defective spermatogenesis but may be secondary to the resultant infertility in some of these patients. Normal levels of FSH in infertile patients with mild to moderate oligospermia and an increase in serum FSH in a patient with severe oligospermia was observed by Saeed *et al.* (10). Significantly higher FSH and LH concentrations were observed in azoospermic men than controls indicating disturbance of the spermatogenic process. However, no significant difference was observed in serum testosterone between azoospermic men and controls . A third of infertile subjects with dyspermia was observed with hyperprolactinemia. In a sub-Saharan African country, 52.9% of men with hormonal disorders with hyperprolactinemia were

observed which was the most common form of hormonal abnormality (7).

In this present study, comparison of the mean concentrations of FSH, LH, prolactin and testosterone between fertile and infertile men showed no significant differences (p.0.05). This indicates that hormonal factors may not be responsible for infertility and the observed defect in semen quality in infertile men may be as a result of some other factors. Similar observations were made in other studies (4,6,12). Similarly, LH, FSH and testosterone were not significantly different between men with STDs and fertile controls ($p>0.05$ Table 1) in this present study. However, prolactin was the only hormone that showed a significantly lower difference in men with STDs than fertile controls ($p=0.03$). Low testosterone and prolactinemia were observed in association with high bacterial and non-bacterial infection (13). The reason for these differences is not known and requires further study. The role of prolactin in male infertility is not clear. However, hyperprolactinemia has been proved to have a negative effect on spermiogenesis and spermatogenesis. The finding also of abnormal prolactin levels as the most common observation in KuKu's study (7) makes it perhaps necessary to elucidate the relationship between STDs, prolactin and male infertility. Similar to the report by kuku (7) we equally suggest that direct hormonal evaluation and identification of patients with abnormal prolactin should be an important part of the workup of infertile males in this sub-region – an uncommon practice at present.

CONCLUSION

Hormonal abnormalities may not be a major contributor to male infertility as no significant differences ($p < \text{or} > 0.05$) were observed in FSH,LH, prolactin and testosterone between infertile and fertile controls.

However, we suggest the inclusion of prolactin evaluation in the routine workup of infertile males because of the significantly lower prolactin levels ($p<0.05$) between the STDs group and fertile controls. This requires further studies.

REFERENCES

1. *Omonria W.E, Egbunike G. N , Ladipo O. A. Classification of the semen of the male partners of infertile Nigerian couples. Andrologia ,1985;17:257-261.*
2. *Marinho AO,Aetiological factors in infertility.A review of 277 infertile Nigerian couples. W.Afri.J. Med.1986;5:69-74.*
3. *Ilesanmi A O. Adeleye, JA ,Osotimehin B O, Comparison of single serum progesterone and endometrial biopsy for confimation of ovulation in infertile Nigerian* women. Afr.J. Med. Sci. 1995; 24: 97-101.
4. *Nkponsong E O, Lawani J, Osanyintuyi SO, Awojobi O A, Semen analysis in infertility in Ibadan. Nig. Med. J. 1982; 12: 181-86.*
5. *Weinbauer G F, Nieschlag E, Gonadotrophin control of testicular germ cell development (Review). Adv. Exp. Med. Biol. 1995; 377:55-65.*
6. *Mcclure RD, Endocrine Investigation and Therapy. Urol. Clin. Nor. Am. 1987; 14: 471-488.*
7. *Kuku S, African endocrine infertility: A review. Afr. J. Med. Sci. 1995; 24:111-123.*
   Arya O P, Osoba AO, Benett FJ, Tropical Venerology. 2nd Ed. Edinburgh, Churchill Livingstone Inc.1988; 370.
8. *Ekweozor CC, Olaleye OD, Tomori O, Saliu I, Essien EM, Bakare RA et al. Clinico-epidemiological patterns of HIV infection in STD patients in Ibadan. Afr. J. Med. Sci.1995; 33:321-327 .*
9. *Saeed S, Khan FA, Rahman SB, Khan DA. Ahmed M, Biochemical parameters in evaluation of oligospermia. JPMA 1994; 44:137-40.*
10. *Merino GS, Carranza-Lira JC, Martinez-Chequer JC, Barahona E, Moran C, Bermudez JA. Hyperprolactinemia in men with asthenozoospermia, oligozoospermia, or azoospermia. Arch. Androl. 1996; 38:201-6.*
11. *Takahashi , Ogawa K, Tobai K, Kojima T, Shida K, Isula M, Sato K. Clinical significance of direct immunobead test to detect antisperm antibody (Japanese) Nippon Sanka Fujinka Gakkai. Zasshi-Acta Obstetrics and Gynaecologica . Jiponica 1992; 44: 779-86.*
13. *Ekwere PD.Immunological infertility among Nigeria men: incidence of circulating antisperm auto-antibodies and some clinical observations: preliminary report. Br. J. Urol. 1995; 76: 366-70.*
14. *Rogoza A, Mierzejewski W, Puzio M. Detection and treatment of hyperprolactinemia in male infertility. Ginekologia Polska 1994; 65:75-9.*