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TRUE CORRECTED SEMINAL FRUCTOSE IN MALE INFERTILITY IN NIGERIANS A PRELIMINARY STUDY

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ABSTRACT

Background: The function of the seminal vesicle is important for fertility. Low level of true corrected seminal fructose has been observed in hypofunction of the seminal vesicles and has been related to male infertility. In Nigeria, published studies on seminal fluid analyses have so far excluded information on seminal fructose.

Objective: To prospectively determine the true corrected seminal fructose values in Nigerians as it may serve as a cheaper and available alternative for assessing seminal vesicle function, and for overall evaluation, in male infertility in Nigeria.

Materials and Method: The subjects included patients and volunteers. Seminal fluid collection and analysis were done using the WHO standards. Serum testosterone was also measured in all subjects. Correlation was tested with the student "t" test and the level significance was $p < 0.05$.

Result: 317 subjects were studied, 20 were azoospermic, 237 were oligospermic, and 60 were normospermic (normal control). Their ages ranged 24-60 years with a mean of 41.55 ± 7.63 years. Mean values of seminal fructose were 154.35 ± 29.52 mg/dl in azoospermics, 487.03 ± 87.45 mg/dl in oligospermics, and 338.03 ± 86.14 mg/dl in normospermics.

The normal range of seminal fructose was determined to be 163.13-512.93mg/dl. Mean serum testosterone level of the subjects was 2.47 ± 0.63 ng/ml, range 1.3-4.1ng/ml.

There was a correlation between true corrected seminal fructose with motile density in normospermics ($r=0.32$), and it was strongly so with oligospermics ($r=0.734$). Serum testosterone was strongly correlated with seminal fructose in all groups ($r > 0.67$).

Conclusion: In our environment, true corrected seminal fructose may be of value in the evaluation of infertility as a biological marker for androgen activity in the reproductive tract, and of seminal vesicular function.

Key Words: True corrected seminal fructose, male infertility

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INTRODUCTION

Seminal fructose is produced in the seminal vesicles and the World Health Organization (WHO) includes its measurement for the assessment of the functions of these glands. The seminal vesicle is important for fertility because its secretion, which constitutes 50% and the last fraction of the ejaculate, is important for semen coagulation, sperm motility, stability of chromatin, and suppression of immune activity in the reproductive tract. Subjects with its hypofunction have low levels of seminal fructose and low sperm motility²

Earlier attempts at the universal use of seminal fructose as a marker of seminal vesicle function was frustrated because of its inverse relationship with the sperm count and lack of correlation with sperm motility which made it inappropriate.

This problem was removed by excluding the influence of sperm count and motility on fructose concentration with the introduction of the "corrected seminal fructose" and "true corrected seminal fructose (TCSF)"^{3,4} The TCSF, defined as $[\log \text{ motile sperm concentration} \times \text{ seminal fructose concentration}]$ has been shown to be a better marker of seminal vesicle function⁴.

In the underdeveloped economy of Nigeria, if estimation of seminal fructose is developed for the evaluation of seminal vesicle function, it is likely to be simpler, available, and more cost-effective for fertility management.

This prospective study aims at studying seminal fructose and the correlations of TCSF values in infertility in Nigerians. It is hoped that this study will arouse consciousness and stimulate interest in this potential factor in the evaluation of male infertility. It will thus serve as a prelude to exploring the available

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options for its use as a cheaper alternative for assessing seminal vesicle function in male infertility. In Nigeria, apart from that of Nkposong *et al*,⁵ published studies on seminal fluid analysis have so far excluded information on seminal fructose.

PATIENTS AND METHODS.

The subjects were men who were generally in good health condition but who presented at, or were referred to, the MacBenson Hospital, Onitsha, for the evaluation of infertility. They included subjects who were referred to, or presented at the Fezi Reference Medical Laboratory, Onitsha for investigations for infertility. These patients were studied over a six-year period, January 2000 to December 2006. There was also a control group of fertile men who were recruited as volunteers. These volunteers must have fathered children within the preceding one year before presentation, and had no history or evidence of current inflammatory conditions of the genital tract, such as sexually transmitted disease and leucocytospermia. These control subjects also had normal seminology, with sperm cell density of above 20 million cells/ml, active motility of above 60%, and abnormal morphology of below 10%.

Semen production was by masturbation or coitus interruptus after abstinence of at least 3 days. Semen analysis was done using the WHO standards¹. For the purpose of this study, the entire specimens were examined within 30 -40 minutes of collection. Those that could not be so examined were excluded from the study.

Seminal fructose was determined by the Seliwanoff's method as adopted from Wendel⁶

Serum testosterone was evaluated in all the patients with the immunoassay method.

Correlation was tested using the Spearman Rank Correlation co-efficient. Significance was tested with the student "t" test and the level of significance was $t < 0.05$.

RESULTS

A total of 317 males who met the criteria for inclusion in the study, were evaluated. These were divided into 3 groups:

1. Normospermic controls (sperm density > 20 million cells/ml) = 60
2. Oligospermic males (sperm density < 20 million cells/ml) = 237
3. Azoospermic males (sperm density = 0 cells/ml) = 20

The ages of the subjects ranged from 24 - 60 years, mean 41.55 ± 7.63 , and median 41 years.

The mean volume of the semen was 2.37 ± 1.8 ml for the normospermic controls, 2.55 ± 2.36 mls for oligospermic men, and 3.11 ± 2.73 mls for the

azoospermic men

The mean value of the seminal fructose concentration was 338.03 ± 86.14 mg/dl for the normospermic controls, 487.03 ± 87.45 mg/dl for the oligospermics and 154.35 ± 29.52 mg/dl for the azoospermics

The statistically determined normal range of seminal fructose in the normal population was 163.13 - 512.93 mg/dl.

Mean testosterone level was 2.47 ± 0.63 ng/ml, range 1.3 - 4.1 ng/ml.

TCSF had no correlation with semen volume in the 3 groups, $r = 0.37297$ in normospermics, $r = -0.015628$ in oligospermics and $r = -0.035$ in azoospermics.

There is a weak inverse correlation between the TCSF and the motile density in normospermics ($r = 0.032$), but strongly so in oligospermics ($r = 0.735$).

Serum testosterone is strongly directly correlated with the TCSF in all the groups ($r > 0.67$)

DISCUSSION

Low levels of TCSF have been observed in hypofunction of the seminal vesicle and since seminal vesicles are androgen dependent, TCSF can be used as a biological marker for androgen activity in the reproductive tract². Normal values of serum testosterone and low values of TCSF have been found in men with azoospermia due to ejaculatory duct obstruction or to congenital absence of the seminal vesicles. One-third of azoospermics have obstruction of the ejaculatory ducts^{7,8}, and 10% of azoospermics have congenital absence of the seminal vesicle⁹.

Our study shows that TCSF correlates strongly with motile density in oligospermia, and with serum testosterone. It also demonstrates lower values of seminal fructose concentration in azoospermia than in oligospermia or normospermia. But seminal fructose concentrations are known to be higher in azoospermic men than in men with oligospermia or normospermia². This lower value in our study may be an indication of a high proportion of obstructions in our azoospermic population. Other parameters assessed were similar to other studies.

Thus in our environment, a combined measurement of TCSF and serum testosterone may allow for a diagnosis of hypoandrogenism, seminal vesicle dysfunction, and obstructive processes in the reproductive tract. Other methods available for such assessments most often are not available and if available, the cost may be prohibitive to the economy of the populace. These other methods include the measurement of the specific protein of the seminal vesicle (MH-5)¹⁰, measurement of protein C inhibitor¹¹, transrectal ultrasound¹², and magnetic resonance imaging¹³.

CONCLUSION

In our environment, TCSF may be of value in the evaluation and management of male infertility in Nigeria.

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