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EFFECT OF BACTERIAL ISOLATES ON THE SEMINAL INDICES OF MEN INVESTIGATED FOR INFERTILITY IN GOMBE

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ABSTRACT

Context: The significant contribution of the male partner to infertility is no longer in doubt. There is however little disagreement regarding the frustrating experience with medical management of male infertility. The effect of infection as a contributor to abnormalities in semen parameters has been reviewed.

Objectives: The study was aimed at identifying male factor contribution to infertility and the influence of bacterial infection on seminal parameters.

Study-design, setting and subjects: All 202 patients were spouses of infertile women who presented to the Gynaecological clinic of the Federal Medical Centre Gombe over a one-year period, from January to December 2001 inclusive.

Results: The density of spermatozoa ranged from 0-844x10^6/ml with a mean of 44.588.5 x10^6/ml. Only 94 (46.5%) of the patients had the reference lower limit normal density of 20 million spermatozoa/ml or more. However, 111 (55%) were normozoospermic for total count with 64 (31.7%) being oligozoospermic. There were bacterial isolates from the seminal fluid of 134 (66.3%) patients that was predominantly accounted for by S. aureus 67.2% (90/134). The seminal fluid produced was 1-8mls, with a mean of 3.11.7mls of which 45 (22.3%) were oligospermic. The mean pH was 7.10.3, in 16 (7.9%) patients the seminal fluid pH was 8-9. There were 27 (13.4%) azoospermic men, in 163 (80.7%) the spermatozoa had normal morphology of 70% or more while 12 (5.9%) men had abnormal morphology of 30% or more i.e. were teratozoospermic. All cases with no abnormal (or normal) morphology were actually azoospermic. There was a significant association between the presence of bacterial isolate in the seminal fluid and oligozoospermia. However the motility, morphology or the count do not seem to be affected by the presence of infection.

Conclusion: Bacterial presence contribute significantly to poor semen quality in our environment. Primary prevention and prompt treatment of urogenital infections could reduce the infectious contribution to male infertility.

Key Words: Urogenital infection, Male infertility, Semen quality.

INTRODUCTION

The desire of men among others, from time immemorial is to have children. When such hopes and desires are dashed, the consequences are predictable. That the male accounts for 20-48% of infertility is a well established fact[1-4], although most men in Africa still feel immune to the problem. For some time barrenness had been blamed solely on women and this has led to delays in seeking care by the men. This is probably due to the mix up between potency and fertility.[5]

PATIENTS AND METHODS

Seminal fluid was obtained from 202 men whose wives presented with inability to conceive after at least one year of regular unprotected sexual intercourse. Standardised WHO procedures were Used to collect, examine and reach a diagnosis (WHO, 1999, 2000). All patients were spouses of women who presented at the infertility clinic of the Federal Medical Centre Gombe over a one year period, from January to December, 2001.Each specimen was taken by masturbation following three days of sexual abstinence and analysed in accordance with WHO guidelines (WHO, 1999).Microscopy, culture and sensitivity were done on all specimen collected. For the purpose of this study, normospermia refers to a sperm volume of at least 2.0mls/ejaculate; while normozoospermia is used to describe a sperm density of at least 20 x 10^6/ml with at least 2mls of ejaculate i.e. count of 40 x 10^6/ejaculate.

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RESULTS

Table 1 shows that 1-8mls of seminal fluid was produced, with a mean of 3.11.7mls of which 45 (22.3%) were oligospermic. The mean pH was

7.10.3, in 16 (7.9%) patients the seminal fluid pH was 8-9. In 35 patients (17.3%) there was no actively motile spermatozoon, in 17 patients (8.4%) the sluggish motility was 40% or more while 66 patients (32.6%) had dead spermatozoa of 40% or more in the ejaculate as shown on Table 2. An analysis of the motility of the spermatozoa revealed that only 78 patients (38.6%) showed 50% of the spermatozoa had active progressive motility, with necro-asthenozoospermia accounting for the commonest motility abnormality in 86 (42.6%) patients.

Table 3 shows that there were 27 (13.4%) azoospermic men. In 163 (80.7%) the spermatozoa had normal morphology of 70% or more while 12 (5.9%) men had abnormal morphology of 30% or more i.e. were teratozoospermic. All cases with no abnormal or no normal morphology were actually azoospermic. When these are excluded, the normal morphology ranged from 10-98%, with a peak teratozoospermia of 90%. The density of spermatozoa in millions/ml ranged from 0-844 with a mean of 44.588.5 as shown on Table 4. Only 94 (46.5%) of the patients had the reference lower limit of normal density of 20 million spermatozoa/ml or more. However, 111 (55%) were normozoospermic for total count with 64 (31.7%) being oligozoospermic. There were bacterial isolates from the seminal fluid of 134 (66.3%) patients that was predominantly accounted for by S. aureus 67.2% (90/134) as shown on table 5. There was a significant association between the presence of bacterial isolates in the seminal fluid and oligospermia. Although this does not seem to have influenced the motility, morphology or the count (Table 6).

Table 1: **Volume and Ph.**

Factors	Frequency	%
1. Volume		
Normospermia (≥2mls)	157	77.7
Oligospermia (<2mls)	**45**	**22.3**
Total	**202**	**100**
Range = 1-8; Mean = 3.1±1.7		
2. pH		
7	186	92.1
8	13	6.4
9	3	1.5
Total	**202**	**100**
Range = 7-9; Mean = 7.2		

Table 2: **Motility.**

Factors	Frequency	%
1. Active motility		
0	35	17.3
1-49	89	44.1
≥50	78	38.6
Total	**202**	**100**
Range = 0-95; Mean = 40.8±29.6		
2. Sluggish		
0	31	15.3
1-49	154	76.2
≥50	17	8.4
Total	**202**	**100**
Range = 0-80; Mean = 18.5±14.6		
3. Dead		
0	29	14.3
1-49	107	53.0
≥50	66	32.7
Total	**202**	**100**
Range = 0-100; Mean = 27.8±26.9		
4. Motility analysis		
Necro-asthenozoospermia	86	42.6 (dead + sluggish =50%)
Normal	79	39.1(at least 50% active)
Azoospermia	27	13.4 (no spermatozoa)
Necrospermia	8	4.0 (at least 50% dead)
Asthenospermia	2	1.0 (at least 50% sluggish)
Total	**202**	**100**

Table 3: **Morphology.**

Factors	Frequency	%
1. Normal		
0	27	13.4
1-69	12	5.9
≥70	163	80.7
Total	**202**	**100**
Range = 0-98%; Mean = 73.6%±31.4%		
2. Abnormal		
0	27	13.4
1-29	163	80.7
≥30	12	5.9
Total	**202**	**100**
Range = 0-90%; Mean = 13.0%±13.0%		
3. Morphology analysis		
Normal	163	80.7
Azoospermia	27	13.4
Teratozoospermia 12	5.9	
Total	**202**	**100**

Table 4: **Density and Count.**

Factors	Frequency	%
1. Density		
0	27	13.4
1-19	81	40.1
≥20	94	46.5
Total	**202**	**100**
Range = 0-844; Mean = 44.5±88.5		
2. Count		
0	27	13.4
1-39	64	31.7
≥40	111	55.0
Total	**202**	**100**
Range = 0-2,110; Mean = 126.0±227.8		
3. Count analysis		
Normozoospermia	111	55.0
Oligozoospermia	64	31.7
Azoospermia	27	13.4
Total	**202**	**100**

Table 5: **Microbial Isolates.**

Factors	Frequency	%
1.Presence of organism		
Yes	134	66.3
No	68	33.7
Total	**202**	**100**
2. Isolates		
S. aureus	90	67.2
Streptoccoci	24	17.9
E. coli	9	6.7
Klebsiella	8	6.0
Proteus	2	1.5
Gonococcus	1	0.7
No growth	68	33.7
Total	**202**	**100**

Table 6: **Effect of Bacterial Isolates on Seminal Parameters.**

Factors	No Bacteria	Bacteria	Total
1. Volume			
Normospermia	60	97	157
Oligospermia	8	37	45
Total	**68**	**134**	**202**
	X^2 = 6.5; P = 0.01		
2. Motility			
Azoospermia	6	21	163
Necro-asthenozoospermia	31	65	96
Normozoospermia	31	48	79
Total	**68**	**134**	**202**
	X^2 = 2.76; P = 0.25		
3. Morphology			
Azoospermia	6	21	27
Normozoospermia	56	107	163
Teratozoospermia	6	6	12
Total	**68**	**134**	**202**
	X^2 = 3.1; P = 0.1		
4. Count			
Azoospermia	6	21	27
Normozoospermia	44	67	111
Oligozoospermia	18	46	64
Total	**68**	**134**	**202**
	X^2 = 4.2; P = 0.1		

DISCUSSION

The recommended investigations for male infertility are beyond our reported findings at microscopy and culture, but also include biochemical and immunological tests that are not available in our centre thus setting a limit to the extent of diagnosis that can be reached. It has been suggested that semen volume is significantly higher in infertile than in fertile couples but its relationship with conception has not been confirmed.[6,7] In this study 77.7% of the patients had semen volume ≥2mls. Oligozoospermia was found in 31.7% of the men in this study. This is lower than the 44.6% reported by Nkposong et al but higher than the 21.0% and 26.8% reported by Imade et al and Idrisa et al repectively.[8,9,18] The high oligozoospermic rate in this study could be due to the high prevalence of urogenital infections. Other possible causes are: physiological, psychological, or immunological origin or even an artefact.[17] The exact determination of a sperm count low enough to warrant complete infertility is difficult: so long as but one spermatozoon is present, the possibility for fertilization exists.[11]

It is recommended that a normal ejaculate should contain 20 x 10^6 spermatozoa/ml or more, giving a total count of 40 x 10^6 spermatozoa/ejaculate or more.[9] Over fifty three (53.5%) had a below normal density hence oligozoospermic, but only 31.7% had a total count below 40 x 10^6 spermatozoa/ejaculate. This implies that some men with low sperm density are able to compensate for their total spermatozoa count through their semen volume. Azoospermia was found in 13.4% of the men studied. This is higher than the 9.1%,[9] and 4% [10] but lower than 35%[8] earlier reported. It is however similar to 12.8% reported from Maiduguri.[18] An analysis of the motility of the spermatozoa revealed that only 79 patients (39.1%) were normozoospermic, with necro-asthenozoospermia accounting for the commonest abnormality of motility in 86 (42.6%) patients. The poor motility could be the result of infection, which was not uncommon in this study even though there was no statistically significant relationship in this study. Endocrine abnormalities and antisperm antibodies which were not evaluated in this study could also contribute to the low motility.[9,12] Motility is temperature-dependent, and could be affected by variations in room temperature from air condition, heating, or open windows.[11] Some studies indicate that the motility of spermatozoa in the semen increases as the count increased but whether this enhances fertility or not is not clear.[9,13] That 5.9% had teratozoospermia against the backdrop of bacterial isolates in 66.3% contrast with the established relationship between infection and teratozoospermia.[8,11,14] perhaps because many of the organisms cultured may be harmless commensals. Exposure to certain drugs that affect spermatogenesis (e.g., nitrofurans, cimetidine) produces an increase in the number of abnormal sperm[15] but an ejaculate is not considered abnormal unless fewer than 15% perfectly normal sperm forms are present.[16]

There were bacterial isolates from the seminal fluid of 134 (66.3%) patients, which was predominantly accounted for by S. aureus 67.2% (90/134). Similar findings have been reported from other parts of Nigeria.[18-20] The organisms were mostly resistant to the commonly prescribed antibiotics. This could explain failure of improvement in semen parameters after adequate antibiotic treatment. It is therefore important to review sensitivity pattern periodically. Like the study from Enugu[19], there was a significant association between the presence of bacterial isolate in the seminal fluid and oligozoospermia in this study (p=0.01). Although this does not seem to influence the motility, morphology or the count which is in contrast to other studies.[8,14,17,19]

CONCLUSION

The findings in this study suggest that bacterial presence contribute significantly to low sperm counts. Primary prevention of urogenital infection and prompt treatment of established infection will therefore reduce the contribution of infection to male infertility in our environment.

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