**Peer Reviewed, Open Access, Free**
Published Quarterly
Mangalore, South India
ISSN 0972-5997
Volume 8, Issue 1; Jan-Mar 2009

**Original Article**

# Contribution of Bacterial Infection to Male Infertility in Nigerians

**Authors**
**Emokpae MA,**
Dept. Of Chemical Pathology, Aminu Kano Teaching Hospital, Kano,
Dept. Of Biochemistry, University of Benin, Benin City
**Uadia PO,**
Dept. Of Biochemistry, University of Benin, Benin City,
**Sadiq NM**
Dept. of Medical Microbiology, Aminu Kano Teaching Hospital, Kano

**Address For Correspondence**
**Emokpae MA,**
Dept. Of Chemical Pathology,
Aminu Kano Teaching Hospital,
P.M.B3452, Kano,
Nigeria.
**E-mail:** biodunemokpae@yahoo.com

**Citation**
Emokpae MA, Uadia PO, Sadiq NM. Contribution of Bacterial Infection to Male Infertility in Nigerians. *Online J Health Allied Scs*. 2009;8(1):6

**URL**
http://www.ojhas.org/issue29/2009-1-6.htm

Submitted: Mar 11, 2009; Accepted: Apr 10, 2009; Published: May 5, 2009

**Abstract:**
There is disagreement as to the influence of certain microbial infection on male infertility and such agents are ignored. The incidence of these microbial agents in seminal fluid isolates is on the increase. This study therefore evaluates the prevalence of male factor infertility and contribution of microbial infection to male infertility in Kano, northern Nigeria. Seminal fluid analysis in five hundred males who were investigated for infertility was evaluated using the 5$^{th}$ generation SQ AII C-P sperm quality analyzer and the Neubaeur counting chamber. The result indicates that 58.2% had sperm density less than twenty million per millilitre. The oligospermic subjects (sperm density 2-19 millions/ml) were 27.6%, severe oligospermic (sperm density less than 2 million) 13.2% and azoospermia, 17.4%. Asthenospermia (motility less than 50%) decrease from 44.8% in oligospermia to 24.0% in severe oligospermia. Teratospermia (abnormal morphology greater than 50%) also deteriorated from 46.3% to 35.4% in oligospermic and severe oligospermic males respectively. Seminal fluid infection increases with decreasing sperm density, motility and morphology. The prevalence of abnormal sperm indices and bacterial infection is high and *Staphylococcus aureus* infection should be treated and no longer ignored in the management of male factor infertility.
**Key Words:** Male infertility, oligospermia, severe oligospermia, azoospermia infection

## Introduction:

There is disagreement as to the influence of certain microbial infection on male infertility. Several investigators have reported difference types of organisms in seminal fluid specimens depending on the methods of examination.[1] It was reported that detection of bacteria in semen does not necessarily suggest infection since bacteria isolates in seminal fluid may represent contamination, colonization of the urethral orifice or infection. *Chlamydia trachomatis, Ureaplasma urealyticum* and enterobacteria are the most frequently isolated organisms in industrialized countries.[2] In easthern and southern parts of Nigeria, oligospermia and azoospermia are the common causes of male factor infertility which has been attributed to bacterial infections.[3-4]

According to the World Health Organisation (WHO)[5], seminal fluid infection was defined as the presence of significant bacteriospermia ($\geq 10^3$ bacteria/ml ejaculate), detection of *Neisseria gonorrhoeae*, *C. trachomatis*, *U. urealyticum*; significant leukocytospermia ($10^6$ peroxidase positive leukocyte/ml ejaculate). It therefore follows that if some or all the conditions above are not met, the isolation of bacteria in semen are often regarded as contaminants by most practitioners. The prevalence of male factor infertility in sub-Saharan Africa is on the increase.[6-7] This study is therefore aimed at determining the prevalence and role of bacteria infection in male factor infertility in Kano, northern Nigeria.

## Material and Methods:

Five hundred seminal fluid specimens from men investigated for infertility over a period of 4years were analyzed. These were seminal fluids of patients referred to the laboratory from the fertility clinics of Aminu Kano Teaching Hospital. The specimen was collected either by self or assisted masturbation into sterile bottle. The subjects were tutored on how to collect the specimens and submit to the laboratory within one hour of production. They were told to first pass urine and then wash their hands and penis with soap, then rinse with water prior to masturbation and ejaculation into sterile container. The semen was collected after the patient had abstained from coitus for at least 3days.

The semen was them cultured on blood and chocolate agar media and incubated for 24hours at $37^oc$. The infective organisms were identified by Gram Stain, coagulase and biochemical reactions and sensitivity to various antibiotic determined. The analysis was done using a standard Neubaeur counting chamber and the $5^{th}$ generation SQAII C-P sperm quality analyzer by medical electronic system, Ltd England. Where the improved neubaeur counting chamber was used, the semen was diluted 1 in 20 with 1% formalin and the spermatozoa counted under the microscope using x40 objective. Appropriate quality control measure was observed as recommended by World Health Organization, (1999). SQAII C-P quality analyzer has to pass self test before analyses was done. The sperm density, volume, viscosity (liquefaction), the percentage of actively motile sperms, the percentage of abnormal forms, the presence or absence of pus cells were evaluated. The semen was analyzed on two different occasions at eight weeks interval for those semen specimens which gave abnormal results. The average of the two readings was calculated. Analysis was carried out immediately they were received. Seminal fluid fructose reaction was carried out on all azoospermic specimens using a mixture of resorcinol/HCl reagent.

## Results:

The results are as summarized in Tables 1,2,3 and 4. Table 1 shows that 291 (58.2%) of the subjects had abnormal seminal fluid sperm density, motility and morphology, while 209 (41.8%) of the subjects had seminal fluid cell density above twenty million per millilitre, motility and morphology above 60% respectively. Whereas oligospermic subject had sperm morphology of 46.3%, motility 44.18% and density of 8.8 x $10^6$ cell/ml, severe oligospermic subjects had sperm morphology of 35.4%, motility of 24.0% and density of 0.63 x $10^6$ cell/ml. Of the two hundred and nine patients whose sperm count was more than twenty million per millilitre, seminal fluid infection was detected in thirty one (14.8%). Seminal fluid infection was detected in fifty-one (35.2%) of oligospermic patients. Similarly infection was detected in twenty six (44.1%) of severe oligospermic subject and sixty five (74.7%) in azoospermic patients (table 2). Table 3 indicates *Staphylococcus aureus* was detected in one hundred and eighteen (68.2%) infected seminal fluids, *Escherichia coli* was detected in thirty one (17.9%) and *Candida species* in ten (5.78%). Other pathogenic organisms detected were mixed growth of Escherichia coli and *Staphylococcus aureus* in eight (4.62%) and *streptococcus species* in six (3.49%) seminal fluids.

Seminal fluid fructose was negative in only one seminal fluid out of the eighty seven azoospermic seminal fluids (Table 4).

**Table 1: Seminal Fluid Analysis (Mean ± SEM)**

| Sperm | Percent(%) | Age (Mean) | Volume (ml) | Morphology (%) | Motility (%) | Density ($X10^6$ cell/ml |
|---|---|---|---|---|---|---|
| **Normospermia (>20x$10^6$ cell/ml) n = 209** | 41.8 | 38.6±1.02 | 2.68.2±0.09 | 68.2±1.26 | 76.1±1.38 | 52.69±2.66 |
| **Oligospermia (2-19x$10^6$ cell/ml) n= 145** | 27.6 | 39.6±1.35 | 2.39±0.16 | 46.3±1.43 P<0.001 | 44.18±1.9 P<0.001 | 8.80±0.46 P<0.001 |
| **Severe Oligospermia (<2x$10^6$ cell/ml) n= 59** | 13.2 | 36.2±1.18 | 2.63±0.15 | 35.4±2.45 p<0.001 | 24.0±2.24 P<0.001 | 0.63±0.06 P<0.001 |
| **Azoospermia (no sperm cells) n=87** | 17.4 | 38.8±2.46 | 2.39±0.16 | - | - | - |

**Table 2: Seminal Fluid Infection and Density**

| Sperm density ( x $10^6$ cell/ml) | Number of patients | Number infected | Percentage |
|---|---|---|---|
| > 20 | 209 | 31 | 14.8 |
| 2 – 19 | 145 | 51 | 35.2 |
| < 2.0 | 59 | 26 | 44.1 |
| Nil | 87 | 65 | 74.7 |

**Table 3: Pathogenic Organisms Isolated From Seminal Fluid**

| Organisms | Number | Percentage |
|---|---|---|
| *Staphylococcus aureus* | 118 | 68.2 |
| *Escherichia coli* | 31 | 17.9 |
| Candida species | 10 | 5.78 |
| Mixed growth of Staphylococcus aureus and Escherichia coli | 8 | 4.62 |
| Streptococcus species | 6 | 3.49 |
| **Total** | **173** | **100** |

**Table 4: Seminal Fluid Fructose Reaction in Azoospermic Patients**

| Result of reaction | Number | Percentage |
|---|---|---|
| Orange to red colour (positive) | 86 | 98.9 |
| Colourless reaction (negative) | 1 | 1.1 |
| **Total** | **87** | **100** |

## Discussion

The prevalence of male factor infertility in Kano was observed to be 40.8%. This is consistent with that of Onwudiegwu and Bako[6], who observed a 46% prevalence in Ife, but lower than 55-93% observed in Enugu, eastern Nigeria by Chukwudebelu[8]. Seminal fluid analysis is a generally accepted method of assessing male fertility potential. Macleod and Gold[9] suggested that men with sperm counts above 20 million/ml or total count above 100 million per ejaculate should be considered fertile. Other investigators have revealed that sperm counts above 10 million or 25 million per ejaculate should be considered normal provided other parameters such as motility and morphology are normal [10-11]. The concept of a minimal sperm count adequate for fertility has generated a lot of arguments since it was introduced in the 1920s. It has been demonstrated that pregnancies was achieved by normal males who had spermatogenesis suppressed to about one million per millilitre as part of male contraceptive study[12-13].

Seminal fluid infection contributed in no small measure to reduced sperm density, asthenospermia and teratospermia (abnormal sperm morphology of greater than 50%) in this study. Interestingly, *Staphylococcus aureus* as causative organism accounted for 68.2% of seminal fluid infection in this study. Again this is consistent with that reported by Okon et al.[7] in Maiduguri, where *Staphylococcus aureus* was isolated from 62.5% of the seminal fluids. Most practitioners dismiss this infection as mere contamination which is assumed to be of no significance. The WHO definition of seminal tract infection does not clearly differentiate between infection, contamination and colonization of the genital tract. Semen that passes through the genital tract is routinely contaminated with Gram positive cocci such as *Staphylococcus, Streptococcus* and *Diphteroids* [14]. It is generally accepted that *Staphylococcus aureus* which are coagulase positive is regarded as pathogenic and should be treated. The presence of this microorganism can no longer be ignored. The longer the infection persist, the greater the damage and loss of germ cells. The rate (percent) of infection increases from normospermic to azoospermic males (table 3). According to Bukharin et al.[15] opportunistic microorganisms cause classical infections of the urogenital tract and subclinical reproductive tract infections. These infections of the seminal fluid lead to decrease in the number of spermatozoa, the suppression of their motility, changes in their morphology and fertilizing capacity. Our result shows that 58.2% of the males had sperm density below 20 million/ml. The sperm morphology deteriorated progressively in oligospermic to severe oligospermic males. In other words it decreased with decreasing sperm density. Asthenospermia (motility less than 50%) was also observed in 58.2% of the study population. The motility decrease from 46.3% in oligospermic to 35.4% in severe oligospermic subjects. All semen characteristics were statistically significantly different ($P<0.001$) when compared with normospermic subjects. Similarly severe oligospermic was compared with oligospermic males and found to be significantly different ($P<0.001$).

Seminal fluid fructose is usually done for all azoospermic cases. Fructose produced in the seminal vesicles is androgen dependent and serves as a source of energy for ejaculated sperm. Fructose is absent in individuals with congenital absence of the vas differentia who have no seminal vesicles and those with bilateral ejaculatory duct obstruction. Fructose was absent in only one seminal fluid in our study.

It is concluded that the prevalence of abnormal sperm cells indices and bacterial infection is high. In the management of male factor infertility *Staphylococcus aureus* should be properly treated and no longer ignored.


## Acknowledgements:

We are grateful to all the staff of fertility clinic, obstetrics and gynecology department of AKTH for referring the patients to the laboratory. We wish to also thank all the staff of urogenital laboratory for their assistance.


## References:


1. Auroux M. Urogenital infection and male fertility. J Gynecol Obstet Biol Reprod (Paris). 1988;17(7):869-875.
2. Keck, C., Gerber-Schafer, C.,Clad, A, Wilhelm, C and Breckwoldt, M. Seminal tract infections: impact on male fertility and treatment options. *Hum Reprod Update*. 1998;4(6):891-903.
3. Ajabor, L.M., Ezimokhai, M and Kadiri, A. Male contribution to subfertility in Benin city, Nigeria. Trop *J Obst Gynaecol* 1981;2:53.
4. Megafu, U. Seminal fluid infection and oligospermia. *Trop J Obst Gynaecol* 1991;9(2):10-12.
5. World Health Organisation. Laboratory Manual for the examination of Human Semen and Sperm Cervical mucus interaction 4th Ed, Cambridge University press, 1999.
6. Onwudiegwu U, Bako A. Male contribution to infertility in a Nigerian Community. *J Obstet Gynaecol* 1993;13(2):135-138.
7. Okon KO, Nwaogwu M, Zailani SO, Chama C. Pattern of Seminal fluid indices among infertile Male partners attending the infertility clinic of University of Maiduguri Teaching

Hospital, Maiduguri, Nigeria. *Highland Med J* 2005;1(3):18–23.
8. Chukwudebelu WO. The male factor in infertility. The Nigerian experience. *Int J Infertility*. 1978;23:238
9. Macleod J, Gold, R. The Male factor infertility and infertility part II spermatozoa counts in 1000 men of known fertility. *J Urol* 1951;66:436–449.
10. Zukerman Z, Rodriguez – Rigau LJ, Smith KD, Steinberger E. Frequency distribution of sperm counts in fertile and infertile males. *Fertil Steril* 1977;28:1310–1313.
11. Emokpae MA. A review of laboratory investigations of Male infertility. *Nig J Med* 1999;8(2):104-107.
12. Barfield A, Melo J, Continho H et al. Pregnancies associated with sperm concentrations below 10 million/ ml in clinical studies of a potential male contraceptive method. Monthly depot medroxy progesterone acetate and testosterone esters. *Contraception*. 1979;20:121.
13. Burris AS, Clark RV, Vantman DJ et al. A low sperm concentration does not preclude fertility in man with isolated hypogonadotropic hypogonadism after gonadotropic therapy. *Fertil Steril* 1988;50:343.
14. Fowler, JE Jr, Mariano M. Difficulties in quantitating the contribution of methral bacterial to prostatic fluid and seminal fluid cultures. *J Urol* 1984;132:471-473
15. Bukharin OV, Kuz'min MD, Ivanov IUB. The role the microbial factor in the pathogenesis of male infertility. *Zh Mikrobiol Epidemiol Immunobiol*. 2000;(2):106-110.