**Ascertaining the Impacts of Bacteriospermia on Basic semen Parameters: A Cross-Sectional Study in Port Harcourt, Nigeria**

ABSTRACT

Background

Bacterial infections of the male genitourinary tracts have been ascribed as being responsible for about 15% of male-factor infertility. Though trailed by some dissention, lots of researchers focused on more investigations to unravel the mode of the impacts of the pathogens over the semen parameters, determine effective treatments and garner more insights due to the immense public health benefits it portends. The intent of this study is thus to ascertain the effects of bacteriospermia on male-factor infertility among adult males in the study area.

Methodology

This facility-based retrospective cross-sectional study, involved the assessment of the laboratory records of men who were being evaluated for infertility at various public and private healthcare facilities within the study period. The records of all persons with complete documentation for semen analysis and culture were included while those without complete documentation were excluded. On the whole records of 187 persons were assessed while 175 where included. The processing of specimens followed standard procedure as stated in the standard operating procedure (SOP) manual of the laboratory.

Results

The evaluation of the laboratory records of seminal fluid analysis for 175 males, indicate the oldest subject was 63 years while the youngest was 26 years; the mean age was 40.99 ± 9.558 years, the median age was 38.00 years and the mode was 40 years. The prevalence of normal semen parameters was as follows: volume, (73.7%) motility, (31.4%) concentration, (63.9%,) and morphology, (62.3%,) The overall prevalence of bacterial pathogens in this study was 48.0%. The most prevalent is *Escherichia coli* (27.4%), followed by *Staphylococcus aureus* (25.0%), *Enterococcus faecalis* (19.1%), *Streptococci* (17.9%), *Acinetobacter baumannii* (3.6%), *Klebsiella pneumoniae* (3.6%), *Pseudomonas aeruginosa* (2.4%), and *Proteus mirabilis* (1.2%)

**Conclusion**

The outcome of this, and those of several other studies, makes imperative, the deployment of appropriate laboratory diagnostic tools in the workup for infertility. Culture and susceptibility testing together with seminal fluid analysis and related tests will help in the detection of etiologic agents in symptomatic and asymptomatic bacteriospermia; as well as determine the appropriate antimicrobial agents for the treatment.

**INTRODUCTION**

Semen analysis occupies a pivotal position in the medical laboratory investigation of male factor infertility. Infertility or subfertility denotes an inability to conceive or have a baby after twelve months of trying to conceive naturally by having regular and unprotected sexual intercourse.1 It is estimated that 10 – 15% of married couples are facing challenges of infertility2, amounting to between 48 million couples and 186 million individuals globally.3 About 50% of infertility cases are attributable to male factors; which include: genetic defects, steroid hormone disorders, hypogonadism, spermatogenesis dysfunction, ejaculation disorders, anatomical defects and infections of the reproductive tracts.4

About 15% of male factor infertility are reckoned as fallouts of acute or chronic microbial infections of the male genitourinary tracts, which induce inflammatory reactions. These engender deleterious consequences on spermatogenesis and sperm cell functions; with resultant impairment of the quality and quantity of the semen.5 Different kinds of microorganisms such as bacteria, viruses, and parasites, infect the male reproductive system. Bacterial pathogens such as *Chlamydia trachomatis*, *Staphylococcus aureus*, *Staphylococcus saprophyticus, Enterococcus faecalis, Neisseria gonorrhoeae*, *Escherichia coli,* Brucella, *micrococci, Streptococcus agalactia,*and alpha-haemolytic streptococci, contribute to male infertility cases by infecting different organs like the urethra, seminal vesicles, prostate, epididymis, vas deferens, and testes where they cause diseases.4,5,6

One of the commonest bacterial isolates from semen is *Escherichia coli,* which is also the most prevalent cause of urinary tract infections. It has been ascribed with impaired sperm motility, and acrosomal function. *Enterococcus faecalis* has been shown to impair sperm concentration and morphology. *Ureaplasma urealyticum* is a frequent cause of male infertility due to its ability to impair motility, density and morphology of sperm cells. It also reduces the oxidoreductive potential of the ejaculate, thus rendering the sperm cell vulnerable to peroxidative damage.5 It is not all bacteria that infects the male reproductive system that causes male infertility. Mycoplasma may however, be transmitted to females where impairment of female fertility may occur.4, 7

One of the commonest conditions associated with male infertility is oligoasthenoteratozoospermia, characterized by defective counts, motility, and morphology of spermatozoa. In addition to factors already listed male subfertility could be idiopathic.7 The organisms associated with bacteriospermia may be part of the microbiota of the male urogenital tract or can be sexually transferred male urogenital microbiota or sexually transmitted organisms.7 Dysbiosis in male urogenital microbiota is a common source of symptomatic and asymptomatic infections which may be a cause of infertility.6,8 Disorders of the endocrine system (usually due to hypogonadism) contributes about 2% to 5% of cases in male-factor infertility; others include obstruction of sperm transport (such as vasectomy) - 5%, primary testicular defects (abnormal sperm parameters without any identifiable cause) - 65% to 80% and idiopathic causes (male-factor infertility where normal semen parameters are normal) - 10% to 20%.These are only estimates, as there are no accurate statistics due to pervasive underreporting, cultural factors, and regional variations.9

The changes in microbiota occasioned by dysbiosis may result in inflammatory reactions, oxidative stress, production of toxins, alterations in spermatogenesis and sperm structural deterioration with resultant adverse effects on semen quality and male fertility.10 The impacts of bacterial infections on semen quality and the study of male fertility have not received much attention in port Harcourt and Nigeria. This study was thus intended to determine the consequences of bacteriospermia on male-factor infertility by assessing the impact of bacterial infections on basic semen parameters.

**METHODS AND MATERIALS**

**Study period and area**

The study was an evaluation of the records of semen analysis and culture results conducted from January 2021 to December 2023, at Diagnostix Medical Laboratories in Port Harcourt, South-South of Nigeria. The facility is dedicated to rendering services to persons attending public and private healthcare facilities within and outside the Port Harcourt metropolis. The laboratory services are managed by a team of medical laboratory scientists led by an experienced medical laboratory scientist, skilled in the analysis of semen and other body fluids.

**Study design**

The study is a facility-based retrospective cross-sectional study involving the assessment of the laboratory records of men who were being evaluated for infertility at various public and private healthcare facilities within the study period. The records of all persons with complete documentation for semen analysis and culture were included while those without complete documentation were excluded. On the whole records of 167 persons were assessed while 156 where included. The processing of specimens was as stated in the standard operating procedure (SOP) manual of the laboratory.

**Semen analysis**

The persons for seminal fluid analysis (SFA) and culture and sensitivity investigations were instructed to abstain from intercourse for 3–7 days prior to collection of samples. The methods of sample collection were masturbation or coitus interruptus directly into sterile plastic specimen container; condoms were not used in collection of the samples. Post collection, the samples were kept at 37 °C and analyzed within 30 and 60 minutes. The methods and standards specified by the World Health Organization (WHO) laboratory manual for the examination and processing of human semen 2010 were followed during the processing of the samples as contained in the standard operating procedure (SOP) manual of the facility. Sperm count was performed microscopically, using a modified Neubauer counting chamber after diluting the semen specimen (1:20) in a semen diluting fluid. Motility was carried out by examination of the wet preparation of the specimen under a microscope. The determination of sperm morphology involved centrifuging the semen samples and making smears from the deposit, then staining with Papanicoloau and hematoxylin and eosin stains, drying and examining under the microscope. The power of hydrogen (pH) value was measured using pH paper and compared accordingly against the accompanying strips10

**Semen Culture**

Semen microscopy and culture were performed within 3 hours of specimen collection by seeding the samples on blood agar and MacConkey agar, using standard wire loops (10 µL diameter)  and incubated at temperature of 370C for 23 hours, following standard microbiological protocols.. Samples with more than 1000 colony forming units were considered as being bacteriospermic. The bacterial isolates were identified following standard protocols.2

**Measurements of Semen Parameters**

The interpretation of the results was in accordance with the 5th edition of the WHO Laboratory Manual for the Examination and Processing of Human Semen released in 2010 by the World Health Organization The following parameters represent the accepted 5th percentile (lower reference limits) and 95% confidence intervals (CIs): volume: 1.5 mL (95% CI = 1.4–1.7); sperm concentration: 15 million spermatozoa/mL (95% CI = 12–16); morphology: 4% normal forms (95% CI = 3–4); vitality: 58% live (95% CI = 55–63); progressive motility: 32% (95% CI = 31–34); and total motility (progressive + non-progressive motility): 40% (95% CI = 38–42).8

**Terminologies and Interpretations of Semen Parameters**

**Volume:** Euzoospermia describes optimal semen volume (1.5 – 5.0ml); Hypozoospermia is below optimal volume (< 1.5ml); Hyperzoospermia is higher than optimal volume (> 5.0ml).

**Progressive Motility:** Normokinesis is normal progressive motility (≥ 32%), asthenozoospermia implies subnormal progressive motility where less than 32% of sperm cells manifest progressive motility. Necrozoospermia implies the all of the spermatozoa are nonviable.

**Concentration (Counts):** Normozoospermia denotes normal counts (15 – 250 million cells per ml of semen), mild oligozoospermia signify low counts (5 to <15 million / ml), severe oligozoospermia mean very low counts (<5 million /ml), polyzoospermia indicate higher than normal counts (> 250 million /ml), Azoospermia connote a complete absence of spermatozoa.

**Morphology:** Normozoomorphia implies normal morphology (normal forms ≥ 4%), teratozoospermia infers abnormal morphology (normal forms < 4%)

**Combinations of abnormalities:** oligoasthenoteratozoospermia refer to abnormalities in all three cellular parameters – motility, concentration and morphology; other combinations include defects in two of the parameters as follows: oligoasthenozoospermia (subnormal concentrations and motility), asthenoteratozoospermia (subnormal motility and morphology), oligoteratozoospermia (subnormal concentrations and morphology) and teratopolyzoospermia (subnormal morphology and higher than normal motility).

**Statistical analysis**

The data were organized and clarified using Microsoft Excel version 16; the analysis was performed using IBM SPSS Statistics version 25. The associations between infections with semen parameters and bacterial isolates were ascertained for significance using Chi square test of independence at 0.05 level of significance

**Ethical considerations**

Written approval was sought for and obtained from the management of Diagnostix and Scientifique laboratories. The study was reviewed and approved by the Ethical review committee of Faculty of Medical laboratory science, Federal university Otuoke, Nigeria.

**RESULTS**

This study was a review of medical laboratory records of 175 males pertaining to seminal fluid analysis carried out between January 2021 and December 2023. The oldest subject was 63 years while the youngest was 26 years; the mean age was 40.99 ± 9.558 years, the median age was 38.00 years and the mode was 40 years. The highest number of subjects constituting 28% were in the 36-40 years age bracket, while the lowest number (8%) were between 26 and 30 years. The pH of all the semen samples were normal, ranging from 7.2 to 7.9, almost all the samples had normal viscosity (98.6%) at the time of collection and liquified within 15-30 minutes. (table 1) The frequencies of the age and basic semen parameters as presented by descriptive statistics are shown in table 1.

**Table 1: Descriptive Statistics of Age and Basic Semen Parameters**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| **Characteristics** | **Mean** | **Median** | **Mode** | **STD Deviation** | **Minimum** | **Maximum** |
| Age (years) | 40.99 | 40.00 | 39 | 7.75 | 26 | 63 |
| Volume (ml) | 2.380 | 2.000 | 2.0 | 1.38 | 0.2 | 8.5 |
| Motility (%) | 27.36 | 23.00 | 5 | 21.97 | 0 | 68 |
| Counts (Millions/ml) | 47.78 | 28.30 | 0.0 | 57.23 | 0.00 | 300.90 |
| Morphology (Normal %) | 9.94 | 7.00 | 3 | 10.29 | 0 | 52 |
| pH | 7.456 | 7.400 | 7.4 | 0.18 | 7.2 | 7.9 |

**Forms and prevalence of Defects found among the Spermatozoa**

The overall prevalence of normospermia in this study was found to be 31%, and the abnormal cells were 69%. Ten spermatozoa defects were identified, the single defects (asthenozoospermia, teratozoospermia oligozoospermia and polyzoospermia) amounted to 30.8%, those with two defects (oligoasthenozoospermia, asthenoteratozoospermia, oligoteratozoospermia and teratopolyzoospermia) were 11.4%, azoospermia was 4.0%, oligoasthenoteratozoospermia constituted 23% of the total samples. The commonest abnormality was asthenozoospermia (26%). (Figure 2)

**Figure 1: Prevalence of Spermatozoan Abnormalities**

**Seminal Volume and the Impacts of Bacteriospermia**

A total of 73.7% of the semen samples were found to be euzoospermia or having normal volumes ≥ 1.5 ml. The hypozoospermia specimens (volumes below the threshold of 1.5 ml) were 22.3%, while hyperzoospermia (volumes above 5.5ml) constitutes 4.0% of the semen specimens. The age bracket, 31-35 had the highest prevalence for hypozoospermia, (34.6%) while 46-50 age bracket were found with the highest prevalence of euzoospermia at 88.2%. Among euzoospermia 73.3% had bacterial growths, while 74.2% were negative cultures. Hypozoospermia samples recorded 24.4% positive cultures and 20.2 negative cultures. Hyperzoospermia had growths in 2.3% and absence of growth in 5.6. All the bacterial strains had in azoospermia semen, two were absent in hypozoospermia samples and only two were present in hyperzoospermia.

**Table 2 Seminal Volume and the Impacts of Bacteriospermia**

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Characteristics** | **Total** | **Euzoospermia 1.5-5.5 ml** | **%** | **Hypozoospermia < 1.5 ml** | **%** | **Hyperzoospermia ≥5.5 ml** | **%** |
| **Volume** |  |  |  |  |  |  |  |
| **Age Brackets** |  |  |  |  |  |  |  |
| 26-30 | 14 | 12 | 85.7 | 2 | 14.3 | 0 | 0.0 |
| 31-35 | 26 | 17 | 65.4 | 9 | 34.6 | 0 | 0.0 |
| 36-40 | 49 | 35 | 71.4 | 12 | 24.1 | 2 | 1.1 |
| 41-45 | 42 | 32 | 76.2 | 9 | 31.4 | 1 | 2.4 |
| 46-50 | 44 | 33 | 75.6 | 7 | 15. | 4 | 9.1 |
| **Culture** |  |  |  |  |  |  |  |
| Growth | 86 | 63 | 73.3 | 21 | 24.4 | 2 | 2.3 |
| No Growth | 89 | 73 | 74.2 | 18 | 20.2 | 5 | 5.6 |
| **Bacteria** |  |  |  |  |  |  |  |
| *Acinetobacter baumannii* | 3 | 1 | 33.3 | 1 | 33.3 | 1 | 33.3 |
| *Enterococcus faecalis* | 16 | 12 | 65.2 | 4 | 25.0 | 0 | 0.0 |
| *Escherichia coli* | 23 | 15 | 65.2 | 7 | 30.4 | 1 | 4.2 |
| *Klebsiella pneumoniae* | 3 | 3 | 100 | 0 | 0.0. | 0 | 0.0 |
| *Proteus mirabilis* | 1 | 1 | 100 | 0 | 0.0 | 0 | 0.0 |
| *Pseudomonas aeruginosa* | 2 | 1 | 50.0 | 1 | 50.0 | 0 | 0.0 |
| *Staphylococcus aureus* | 21 | 16 | 76.2 | 5 | 23.8 | 0 | 0.0 |
| *Streptococci* | 15 | 12 | 80.0 | 3 | 20.0 | 0 | 0.0 |
| No Bacteria | 91 | 68 | 74.2 | 18 | 19.9 | 5 | 2.9 |
| **Total** | **175** | **129** | **73.7** | **39** | **22.3** | **7** | **4.0** |

**Total Seminal Motility and the Impacts of Bacteriospermia**

Overall, the prevalence of neurokinesis (progressive motility of ≥32%) i.e. optimal motility was 31.4%; asthenozoospermia (progressive motility less than 32%), 53.7%, necrozoospermia (all cells were dead) 4.0%, and azoospermia, 4.6% Within the age brackets, the highest prevalence of neurokinesis (50.0%) was observed with the 26–30-year-olds, while the highest prevalence of asthenozoospermia (60.5%) was observed within the 31-35-year-olds. Neurokinesis specimens were found to have 39.3% of sterile cultures and 36.0% of the positive cultures. Seven of the eight bacterial species were found in neurokinesis semen, asthenozoospermia (six), necrozoospermia (one) and azoospermia (two).

**Table 3 Total Seminal Motility and the Impacts of Bacteriospermia**

|  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Characteristics** | **Total** | **Neurokinesis** | **%** | **Asthenozoospermia** | **%** | **Necrozoospermia** | **%** | **Azoospermia** | **%** |
| **Motility** |  |  |  |  |  |  |  |  |  |
| **Age Brackets** |  |  |  |  |  |  |  |  |  |
| 26-30 | 14 | 7 | 50.0 | 6 | 2.9 | 1 | 7.1 | 0 | 0.0 |
| 31-35 | 26 | 8 | 30.8 | 17 | 65.4 | 0 | 0.0 | 1 | 1.2 |
| 36-40 | 49 | 17 | 34.7 | 26 | 53.1 | 3 | 6.1 | 3 | 6.1 |
| 41-45 | 42 | 17 | 40.5 | 21 | 50.0 | 2 | 4.8 | 2 | 4.8 |
| >45 | 44 | 17 | 38.4 | 24 | 54.5 | 1 | 2.3 | 2 | 4.5 |
|  |  |  |  |  |  |  |  |  |  |
| **Culture** |  |  |  |  |  |  |  |  |  |
| Growth | 84 | 31 | 36.0 | 51 | 59.3 | 2 | 2.3 | 2 | 2.3 |
| No Growth | 91 | 35 | 39.3 | 43 | 48.3 | 5 | 5.6 | 6 | 6.7 |
|  |  |  |  |  |  |  |  |  |  |
| **Bacteria** |  |  |  |  |  |  |  |  |  |
| *Acinetobacter baumannii* | 3 | 0 | 0.0 | 3 | 100 | 0 | 0.0 | 0 | 0.0 |
| *Enterococcus faecalis* | 16 | 5 | 31.3 | 10 | 62.8 | 1 | 6.3 | 0 | 0.0 |
| *Escherichia coli* | 23 | 8 | 34.8 | 14 | 60.9 | 0 | 0.0 | 1 | 4.3 |
| *Klebsiella pneumoniae* | 3 | 2 | 66.7 | 1 | 33.3 | 0 | 0.0 | 0 | 0.0 |
| *Proteus mirabilis* | 1 | 1 | 100 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| *Pseudomonas aeruginosa* | 2 | 1 | 50.0 | 0 | 0.0 | 0 | 0.0 | 1 | 50 |
| *Staphylococcus aureus* | 21 | 6 | 28.6 | 15 | 71.4 | 0 | 0.0 | 0 | 0.0 |
| *Streptococci* | 15 | 7 | 46.7 | 8 | 53.3 | 0 | 0.0 | 0 | 0.0 |
| No Bacteria | 91 | 36 | 39.6 | 43 | 47.3 | 6 | 6.6 | 6 | 6.6 |
| **Total** | **175** | **66** | **37.7** | **94** | **53.7** | **7** | **4.0** | **8** | **4.6** |

**Seminal Counts and the Impacts of Bacteriospermia**

The prevalence of normozoospermia (counts of ≥ 15.0 million/ml), or normal sperm counts, were 63.9%, severe oligozoospermia (counts of < 5.0 million/ml) 12.6%, mild oligozoospermia (counts of 5 - 14.9 million/ml) 17.7%, oligospermia 2.3%, and azoospermia, 4.6%. The > 45 years age bracket recorded the highest normozoospermia of 70%, while the 41-35 bracket. had highest prevalence of 18.4% for the severe oligozoospermia. There were more negative cultures (64.0%) than positive cultures (62.8%) among normozoospermia specimens. Five bacterial strains were isolated from normozoospermia semen; severe oligozoospermia (five), mild oligozoospermia (four), polyzoospermia (one) and azoospermia (two).

**Table 4 Seminal Counts and the Impacts of Bacteriospermia**

|  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| **Characteristics** | **Total** | **Normozoospermia 15.0 – 250 million/ml** | **%** | **Severe Oligozoospermia <5.0 million/ml** | **%** | **Mild Oligozoospermia 5.0 -14.9 million/ ml** | **%** | **Polyzoospermia > 250 milli0onl/ml** | **%** | **Azoospermia** | **%** |
| **Counts** |  |  |  |  |  |  |  |  |  |  |  |
| **Age Brackets** |  |  |  |  |  |  |  |  |  |  |  |
| 26-30 | 14 | 8 | 57.1 | 3 | 21.4 | 2 | 14.3 | 1 | 7.1 | 0 | 0.0 |
| 31-35 | 26 | 14 | 53.8 | 3 | 11.0 | 8 | 30.8 | 0 | 0.0 | 1 | 3.8 |
| 36-40 | 49 | 32 | 65.3 | 5 | 10.2 | 9 | 18.4 | 0 | 0.0 | 3 | 6.14 |
| 41-45 | 42 | 26 | 65.3 | 8 | 18.4 | 6 | 14.3 | 0 | 0.0 | 2 | 4.8 |
| >45 | 44 | 31 | 70.5 | 3 | 6.8 | 6 | 13.6 | 2 | 4.5 | 2 | 4.5 |
|  |  |  |  |  |  |  |  |  |  |  |  |
| **Culture** |  |  |  |  |  |  |  |  |  |  |  |
| Growth | 84 | 54 | 62.8 | 13 | 15.1 | 14 | 16.3 | 3 | 3.5 | 2 | 3.3 |
| no growth | 91 | 57 | 64.0 | 19 | 10.1 | 17 | 19. | 0 | 0.0 | 6 | 6.7 |
|  |  |  |  |  |  |  |  |  |  |  |  |
| **Bacteria** |  |  |  |  |  |  |  |  |  |  |  |
| *Acinetobacter baumannii* | 3 | 2 | 66.7 | 1 | 33.3 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| *Enterococcus faecalis* | 16 | 10 | 62.5 | 2 | 12.5 | 2 | 12.5 | 2 | 12.5 | 0 | 0.0 |
| *Escherichia coli* | 23 | 16 | 69.6 | 2 | 8.7 | 4 | 17.4 | 0 | 0.0 | 1 | 4.3 |
| *Klebsiella pneumoniae* | 3 | 3 | 100 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| *Proteus mirabilis* | 1 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 |
| *Pseudomonas aeruginosa* | 2 | 1 | 50.0 | 0 | 0.0 | 0 | 0.0 | 0 | 0.0 | 1 | 50.0 |
| *Staphylococcus aureus* | 21 | 11 | 52.4 | 4 | 19.0 | 6 | 28.6 | 0 | 0.0 | 0 | 0.0 |
| *Streptococci* | 15 | 10 | 66.7 | 3 | 20.0 | 2 | 13.3 | 0 | 0.0 | 0 | 0.0 |
| No Bacteria | 93 | 58 | 63.7 | 9 | 9.9 | 17 | 18.8 | 1 | 1.1 | 6 | 0.0 |
| **Total** | **175** | **111** | **63.9** | **22** | **12.6** | **31** | **17.7** | **4** | **2.3** | **8** | **4.6** |

**Seminal Morphology and the Impacts of Bacteriospermia**

The overall prevalence of normozoospermia (normal cell morphology ≥ 4%) were 62.3%, while teratozoospermia (cells with normal morphology < 4%) were 32.6%, azoospermia 5.1%. The normozoospermia ranged from 54.4% (41-45-year-olds) to 69.4% (36-40 bracket). There were bacterial growths in 64.0%, and absence of growths in 60.7% of normozoospermia semen. All eight isolates were found in normozoospermia semen; teratozoospermia (five) azoospermia (three).

**Table 5 Seminal Morphology and the Impacts of Bacteriospermia**

|  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- |
| **Characteristics** | **Total** | **Normozoospermia**  **(≥ 4%)** | **%** | **Teratozoospermia (<4%)** | **%** | **Azoospermia** | **%** |
| **Morphology** |  |  |  |  |  |  |  |
| **Age Brackets** |  |  |  |  |  |  |  |
| 26-30 | 14 | 9 | 64.3 | 5 | 35.7 | 0 | 0.0 |
| 31-35 | 26 | 14 | 63.8 | 11 | 42.3 | 1 | 3.8 |
| 36-40 | 49 | 34 | 69.4 | 12 | 24.5 | 3 | 6.1 |
| 41-45 | 42 | 23 | 54.4 | 16 | 38.1 | 3 | 7.1 |
| **≥** 45 | 44 | 29 | 65.9 | 13 | 29.5 | 2 | 4.5 |
|  |  |  |  |  |  |  |  |
| **Culture** |  |  |  |  |  |  |  |
| Growth | 86 | 55 | 64.0 | 28 | 32.6 | 3 | 3.5 |
| No Growth | 89 | 54 | 60.7 | 29 | 32.6 | 6 | 6.7 |
|  |  |  |  |  |  |  |  |
| **Bacteria** |  |  |  |  |  |  |  |
| *Acinetobacter baumannii* | 3 | 2 | 66.7 | 1 | 33.3 | 0 | 0.0 |
| *Enterococcus faecalis* | 16 | 8 | 50.0 | 8 | 50.0 | 0 | 0.0 |
| *Escherichia coli* | 23 | 17 | 73.9 | 5 | 21.7 | 1 | 4.3 |
| *Klebsiella pneumoniae* | 3 | 2 | 66.7 | 1 | 33.3 | 0 | 0.0 |
| *Proteus mirabilis* | 1 | 1 | 100 | 0 | 0.0 | 0 | 0.0 |
| *Pseudomonas aeruginosa* | 2 | 1 | 50.0 | 0 | 0.0 | 1 | 50.0 |
| *Staphylococcus aureus* | 21 | 11 | 52.4 | 10 | 47.6 | 0 | 0.0 |
| *Streptococci* | 15 | 11 | 73.3 | 0 | 0.0 | 1 | 0.6 |
| No Bacteria | 91 | 56 | 61.3 | 6 | 31.9 | 6 | 6.6 |
| **Total** | **175** | **109** | **62.3** | **57** | **32.6** | **9** | **5.1** |

**Prevalence of Bacteriospermia and associated Etiologies**

The overall prevalence of bacterial pathogens in this study was 48.0%. The most prevalent is *Escherichia coli* (27.4%), followed by *Staphylococcus aureus* (25.0%), *Enterococcus faecalis* (19.1%), *Streptococci* (17.9%), *Acinetobacter baumannii* (3.6%), *Klebsiella pneumoniae* (3.6%), *Pseudomonas aeruginosa* (2.4%), and *Proteus mirabilis* (1.2%)

**Figure 2: Prevalence of the Etiologic Agents of Bacteriospermia**

**DISCUSSION**

This study substantially explored the impacts of bacterial infections on semen quality and implicitly on male fertility, which have received little or no attention in the area of study. Bacteriospermia was noticed considerably in semen with normal and abnormal parameters; with 69% of the semen specimens at least one abnormal parameter. The most prevalent of the defective parameters are asthenozoospermia, oligoasthenozoospermia, oligoasthenozoospermia, azoospermia, teratozoospermia and asthenoteratozoospermia. The prevalence of abnormal semen analysis obtained here is a bit higher than 64.71%, 2 reported in an Egyptian study but lower than 84%8 reported in Ethiopia, and 81.17% in India.12 The commonest abnormalities align closely with those in some studies elsewhere2,8,12,

The progressive seminal motility was the most impacted with abnormality, having 31.4% optimal progressive motility (euzoospermia), while 69.6% of the semen specimens were impaired motility (asthenozoospermia), dead/ non-motile cells (necrozoospermia) or azoospermia, where there were no sperm cells in the ejaculates. While 36.0% of normally motile cells were found in semen samples with positive cultures, 39.3% among negative cultures. Though this may appear to indicate a negative effect of bacteriospermia on motility, it was not found significant in this study at 95% CI. While this compares closely with some sudies,17 it is inconsistent with some others.7 Motility appears to be the semen parameter that is most adversely impacted by bacteriospermia. It has been reported that spermatozoa may be immobilized in the presence of microoganisms.12 This has been attributed to a number of mechanisms linked with the presence of bacteria in the genitourinary tracts, such as inflammatory processes, which may cause damages to sperm cells or constrict the passage ducts and impede the sperm motility and seminal flow.13

Negative impacts of bacteria on sperm parameters many be direct or indirect. Direct effect may involve the action of toxins produced by pathogens that may be harmful to the sperm cells.6.7 Bacteriospermia is linked with a number of disease conditions in the male genitourinary tract such as prostatitis or epididymitis, urethritis, and orchitis or damage of the accessory glands which may adversely affect sperm motility and other parameters.6,13 bacteriospermia may negatively impact on motility and other seminal parameters include through cellular interactions, sperm adhesion, agglutinations and related interactions. Also, bacteriospermia in conjunction with leukocytospermia may through the induction of cytokines and ROS generation have negative effects on semen parameters.14,15 Impeding the active and progressively motile sperm cells is probably the precursor to other forms of damage to sperm cells like morphology and concentration.

The overall abnormal semen counts of 36.1% in this study align closely with the findings in some other studies.7 However there appear to be not much disparity in the semen concentration in the semen samples having bacterial growth and those without growths. This is at variance with the findings in some other studies where significant differences were reported,7,13 but align with other studies where no significant differences on the basis of presence or absence of bacteriospermia.2,18 This study did not find any significant impact of bacteriospermia on the semen volume and morphology. This corelates with the findings in some similar studies,2,17 but did not compare well with others.5

The bacterial strains recovered from this study aligns closely with those recovered from aerobic cultures elsewhere.2,5,7,12, The 49.1% prevalence of bacteria as noticed in this study fall with the range of disparate reports from several studies, including the 21% reported in an Italian study,19 25.3% in Nepal,20 35.3% in India,12 34.88% in Iran,7 70.2% in Italy,5 56% in India,21 30% in Saudi Arabia,22 The dominant bacterial isolates in this study were *Escherichia coli Staphylococcus aureus, Enterococcus faecalis and Streptococci..* This was consistent with the findings in studies elsewhere2,7,12, among several other studies

In spite of several research findings supporting the deleterious impacts of bacterial infections on the quality of sperm, the exact dimensions of the impacts remain controversial and yet to be fully unraveled. Studies on antimicrobial treatment of infection-related infertility considers asymptomatic genitourinary infection a major cause of male infertility, which is treatable with antimicrobial agents.7,23 Yet a number of other studies could not find sufficient statistical evidence to corroborate the existence of negative associations between bacterial infections and male-factor infertility.12,16,25 While there were copious presumptive reasons to believe that bacteriospermia exerts negative impacts on semen parameters occasioning infertility, these could not be ascertained at the gross level against bacterial growths in general.

The existence of significant statistical association at 95% CI was found in this study at five instances, namely progressive motility / *Pseudomonas aeruginosa* (p value = 0.016), morphology / *Pseudomonas aeruginosa* (p value = 0.013), *Counts* / *Pseudomonas aeruginosa* (p value = 0.043), counts and *Enterococcus faecalis* (p value = 0.011), and volume and *Acinetobacter baumannii* (p value = 0.025). It can thus be asserted that sufficient statistical evidence is available to corroborate previous findings bacterial infections exert negative impacts on semen parameters which results in male-factor infertility.26-28

*Enterococcus faecalis* is a dominant member of the human microbiome and the semen microbiota, and is among the commonest pathogens associated with bacteriospermia in fertile and sub-fertile men. 12,27-32   *Pseudomonas aeruginosa* and *Acinetobacter baumannii* are also commonly recovered from semen cultures of fertile and sub-fertile men. *Pseudomonas aeruginosa* has been linked impairment of semen parameters.33,34 Pseudomonas-infected semen was reported to be linked with high numbers of oligoasthenoteratozoospermia than controls samples, possibly due to the organism having negative impact on the semen.19 Wile the findings here corroborates previous reports on *Enterococcus faecalis and Pseudomonas aeruginosa,* there were not much reports on the impacts of *Acinetobacter baumannii.*

The limitations of this study include reliance on secondary data, fraught with incompleteness and lack of independence in study design particularly with respect to choice of sociodemographic variables and test methods. The study would have benefited from nucleic acid amplification tests with the advantage of better sensitivity for detecting lower microbial loads commonly associated with asymptomatic infections are recommended for better outcomes.24 The use of Multiplex PCR assays would have conferred more advantages, particularly in cases of co-infections of multiple pathogens, and pathogens that are not easily culturable or differentiate between closely related strains.25

**Conclusion**

Some of the basic semen parameters in this study, namely volume, progressive motility, counts and morphology were apparently impacted adversely by the presence of bacterial infections, however, no significant statistical associations were found at 95% CI to buttress the existence of such associations. The statistically significant association between *Enterococcus faecalis* and defects in semen concentration and morphology is however an apt corroboration of several authorities who reported on the negative effect of bacterial infections on sperm quality.

The outcome of this, and of several other studies, make imperative, the deployment of appropriate laboratory diagnostic tools in the workup for infertility. Culture and susceptibility testing together with seminal fluid analysis and related tests will go a long way in the detection of etiologic agents in symptomatic and asymptomatic bacteriospermia; as well as establish appropriate antimicrobial agents for the treatment. A comprehensive exposition of the role of various pathogens in male infertility will go a long way in dispelling the controversies over this important issue and lead to improved outcomes in infertility management. We could not find any related study within the area, so this study may serve as baseline for further studies in the study area.

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