

Male infertility in Nigeria: A neglected reproductive health issue requiring attention

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ABSTRACT

Even though infertility is not lethal, it has been described as a radical life changing problem that carries with it significant psychological trauma. Male factor infertility is responsible for about 40–50% of all infertility cases. Despite its high prevalence in Nigeria, not much effort has been made at tackling the problem. The impact of male factor infertility is likely to increase if adequate measures are not taken. This paper reviews the main factors that are responsible for the problem and also highlights the need to focus on prevention and management; how those affected could be assisted by government, agencies and the private sector. Internet search of studies on male infertility was done, and those relevant for this study were reviewed. The major causes of infertility in Nigeria are sexually transmitted infections and hormonal abnormalities. Effort should be made in arriving at a proper diagnosis, and adequate treatment given where causes are treatable. Otherwise the patients should be adequately counseled. In irreversible cases, assisted reproductive techniques may be suggested. This procedure as at now is beyond the reach of the average Nigerian citizen. Centers, where such facilities are available, may be subsidized by the government to reduce the cost.

KEY WORDS: Adoption, assisted reproductive techniques, bacterial infection, erectile dysfunction, hormonal abnormalities, male infertility

INTRODUCTION

Infertility refers to the inability of couples to achieve conception despite regular unprotected sexual intercourse for 1-year. It also includes the inability to carry a pregnancy to the delivery of a live baby.^[1] Infertility in the male, therefore, refers to the inability of a man to impregnate a woman after 12 months of regular and unprotected sexual intercourse. That is if the woman has no gynecological problems.^[2] The World Health Organization in 1991 estimated that, 8–12% of couples worldwide experienced some forms of infertility during their reproductive lives, thus affecting 50–80 million couples with 20–35 million in Africa.^[3] It was therefore extrapolated that 3–4 million Nigerian couples were affected.^[4,5] This review highlights the burden and the major causes of infertility in Nigeria and also focuses on the need for preventive measures and helping those with irreversible cases with assisted reproductive techniques.

Methods of literature search

Pubmed and google scholar were used to search for literatures on male infertility, burden and major causes. A total of 2,170,510 articles were initially obtained. The required articles were retrieved between November 2013 and February 2014, and 94 articles were included in the review.

BURDEN OF INFERTILITY IN NIGERIA

Even though infertility is not life threatening, it has been described as a radical life changing problem because it carries with it significant psychological trauma.^[6] The prevalence of infertility in Sub-Sahara Africa ranges from 20% to 40%. Although the Africa socio-cultural setting has before now focused on the female, fertility problems are shared by both male and female sexes. Male factor is responsible for 40–50% of all infertility in Nigeria although it varies from one region to another, and the causes also vary from place to place. A study in the mid-western Nigeria in 2002 showed that about 50% of the 780 couples evaluated were observed to have varied

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causes of infertility.^[7] In the southwest, male factor was reported to be responsible for 42.4% infertility cases,^[8] while in Maiduguri, North-Eastern Nigeria, infertility is the reason for about 40% of all gynecological consultations.^[9] In Kano, 40.8% prevalence was reported,^[10] 46% in Ile-Ife^[11] and 55–93% was observed in Enugu,^[12] Eastern Nigeria for male factor infertility.

Despite the high prevalence of infertility in Africa and Nigeria, in particular, no significant efforts have been made in tackling the problem. The impact of male factor infertility is likely to increase if adequate measures are not taken. In the past, it was even assumed that infertility in Africa did not warrant specific intervention since many African countries have high fertility rate. There were reports that infertility was on the decline in Nigeria.^[13] Data from World Fertility Survey and Demographic and Health Survey showed that levels of infertility appeared to be either declining in Cameroon, Nigeria, and Sudan or remaining stable in Ghana, Kenya and Senegal. The report concluded that infertility has indeed declined among all age groups younger than 40 years by 7.3–6.0 in Cameroon and from 5.6 to 4.2 in Nigeria.^[13] The need for conceptual rethink of infertility in Africa was advocated at the international conference on population and Development in Egypt. Evidence of adverse consequences of infertility on reproductive health is increasingly emerging.^[14,15] Most of the intervention by government and agencies are directed at communicable diseases, and tangible effort is not being made to address the root causes of male factor infertility. The government and developmental partners are mainly concerned with population control and growth.^[16] There is urgent need to focus on preventing infertility and assisting individuals and couples who are already affected by infertility. Management of infertility is not within the scope of many public health policies in Sub-Saharan Africa.^[17] It appears that managers and formulators of public health policy have neglected the problem of infertility for other health issues that are considered to be more urgent and important,^[15] this fact was supported by studies that observed that researchers have concentrated on fertility behavior rather than on infertility.^[18] Akinloye and Truter^[15] suggested recently that since infertility management is morally and logically a low priority for public funding in Nigeria on macro-ethical groups, it should be left to the private sector. They also advocated a national policy that will empower and create a more conducive operating environment for private sector to be involved which may be beneficial to all parties. Public health care delivery should focus on prevention of infertility as one strategy to reduce the number of couples suffering from infertility.

Other reasons attributed to the neglect of male infertility was the dearth of tracking systems in the 20th century to assess the simplest measures of male reproductive

health.^[19] Most of the time, emphasis was placed on female reproduction rather the male. It was assumed that the role men play in human reproduction was elementary hence the sexual organ was likened to a ‘mechanical instrument’ which is either working or not working. While women were considered as primary in reproduction, hence problems of fertility were typically assumed to be female in origin.^[19] Whereas the field of gynecology has been growing, there was little or no growth recorded in the field of andrology. It is only recently that andrology was established as a medical specialty devoted to the study of male fertility.^[19]

MAJOR CAUSES OF INFERTILITY

The etiology of male infertility is multifactorial. This could be genetic, physical abnormalities, injuries, drugs, infections of the genital tract, radiation, toxins or unexplained. The major causes of male factor infertility in Nigeria are infection and hormonal abnormalities.^[10,20,21] Other studies have focused on the contributions of environmental factors, such as diet and toxic elements, cultural behaviors, and genetic factors.

Impact of sexually transmitted diseases

Sexually transmitted infections (STIs) are common problems that are associated with male factor infertility. A study conducted to identify potential risk factors for male infertility in southern Nigeria indicated that there were associations between male infertility and previous exposures to sexually transmitted diseases, unorthodox medication (native medication) and moderate to heavy alcohol consumption.^[7] Men who reported having repeated episodes of penile discharge, painful micturition, genital ulcers and testicular pain were more likely to be infertile.

Another study of a group of 500 males investigated for infertility in Kano, Northern Nigeria, reported 40.8% prevalence of male factor infertility.^[22] The organisms isolated were *Staphylococcus aureus*, *Escherichia coli*, *Candida* species, the mixed growth of *S. aureus* and *E. coli* and *Streptococcus* species.^[23] Seminal fluid infection contributed in no small measure to reduced sperm density, asthenospermia, and teratospermia. *S. aureus* that contributed the most to seminal fluid infection has always been dismissed by most practitioners as mere contamination, hence of no significance. It was concluded in that report that in the management of male factor infertility, this micro-organism should be treated and no longer ignored.^[23] The study also observed that the rate (percent) of infection increased from normospermic 14.8%, oligospermic 35.2%, severe oligospermic 44.1%, to azospermic 74.7%. These infections of seminal fluid often lead to a decrease in the number of spermatozoa and the suppression of their fertility capacity. Microbial infection of the seminal fluid was observed to be the major cause of male infertility. Stephen *et al.*^[24] earlier

observed that the viability and structural integrity of the semen lie on its characteristic feature of mobility. The negative influence of pathogenic micro-organisms on sperm cells' reproductive potential has been observed.^[25] Evidence that *Chlamydia trachomatis*, *Ureaplasma urealyticum* affect fertility and that *Mycoplasma hominis* cause tail abnormalities of spermatozoa abound. The five factors that contribute to overall sperm quality such as sperm motility, speed, density, morphology (shape and size) and liquefaction are all affected by STIs.^[10,26] In a study in Sweden, involving 244 couples treated for infertility, it was observed that a fifth of the men tested positive for antibodies to *Chlamydia*. It was observed that a high number of the patients with antibodies had an ongoing infection.^[27]

Impact of environmental toxins, pesticides, and lifestyle

Human reproductive health may be affected by a number of environmental and occupational exposures to biological (viruses), physical (radiation) and toxic (chemical) sources.^[28] Even though the human body defense mechanisms can protect itself from these harmful sources, these threats are capable of impacting negatively on one's health through ocular or dermal contact, inhalation, ingestion and vertical or horizontal transfer.^[29]

Air pollution

This refers to the release of pollutants such as carbon monoxide, sulfur dioxide, nitrogen dioxide etc., into the atmosphere from various sources such as vehicular exhaust, industrial emission, burning of coal and woods.^[30] Studies have reported that men who are exposed to higher levels of air pollutants were likely to experience abnormal sperm parameters and increased chance of DNA fragmentation.^[31]

Toxic metals

Toxic (heavy) metals such as lead, mercury, cadmium, arsenic and aluminum have been reported to impact negatively on male fertility. Lead that is present in batteries, metal products, pipes, paints and ceramics can interrupt the hypothalamic-pituitary axis and hence alters hormone levels^[29] and spermatogenesis. Mercury is commonly found in thermometers, batteries, and industrial emissions. It is concentration increases in the food chain leading to bioaccumulation that can negatively affect reproductive health.^[29] Mercury can alter spermatogenesis and disrupts fetal development.^[32] We earlier reported increased cadmium levels in seminal plasma and association with sperm parameters.^[33]

Pesticides

Pesticides and other chemicals used in both farming and industries have damaging effects on the reproductive health. They mimic natural hormones, disrupt normal hormone

function and regulate the function of the endocrine system.^[28] Subjects who are occupationally exposed to these chemicals may have the difficulty of reproduction while on the job. Toxic metals and pesticides have negative side effects on those who work with and around them. High levels of common pesticides have been reported in the urine of men who work in agricultural regions and greenhouses, and more than 6% decrease in sperm parameters were also observed in the subjects.^[34]

Organochlorine insecticides such as hisdane have estrogenic activity, and exposure could cause gynaecomastia.^[35] Much of the studies followed the observation that the pesticide dibromochloropropane caused an epidemic of infertility in workers involved in its manufacture, 13% of the exposed group was azoospermic, and another 30% were oligospermic. For the substances that have been investigated in humans, the mechanism in most cases involved a direct testicular toxin rather than suppression of the hypothalamic-pituitary axis. In those studies, serum follicle stimulating hormone (FSH) levels were normal unless azoospermia results at which time FSH can then be elevated.^[35] Some animal studies suggested that toxins's effect on testicular function was mediated by hypothalamic dysfunction. Toxins may act at many stages of sperm development causing abnormal sperm count, motility, and morphology.

Life style

The role that life style plays in the development of infertility has generated a considerable amount of interest. These are the modifiable habits and ways of life that can influence human health and fertility. Many life style factors such as age at which one starts a family, nutrition, body weight, and substance abuse can impact adversely on fertility.^[28]

Age

The age at which a person starts a family may affect fertility in both males and females because fertility peaks and decreases with increasing age. In males, the levels of testosterone decrease as men age and semen parameters deteriorate progressively as from 35 years.^[36-38] After 40 years of age, men have significantly more DNA damage in the semen as well as poor sperm indices.^[39] It was reported that when men are above 45 years their partners stand some risk of delay in time to achieve pregnancy.^[40]

Nutrition

Consumption of a diet rich in carbohydrates, fiber, folate, and lycopene as well as fruits and vegetables correlate with improved semen quality.^[41] Food rich in antioxidants are potentially beneficial for fertility while the consumption of diets high in proteins and fats were reported to impact negatively on fertility.^[26] Antioxidants play important roles in the body by scavenging reactive oxygen species (ROS)

which are produced as by-products of cellular respiration. The endogenous antioxidants present in the body help to eliminate or prevent their harmful effect, but over production of these ROS in the absence of adequate amount of antioxidants results in oxidative stress which may negatively impact on sperm function (motility, decreasing membrane integrity and DNA damage).^[42] Cocuzza *et al.*^[43] observed that consumption of the high amount of antioxidants increased semen quality compared to low amount.^[43]

Body weight

Body weight may impact on reproductive health and is often associated with eating habits and the amount of activity. Body mass index (BMI) is used to describe one's body weight and is calculated by dividing weight (kg) by height (m²). BMI less than 18.5 or above 25 is regarded as underweight or overweight while above 30 is considered as obese.^[44] It was reported that an increase in BMI in the male by ≥ 3 units might correlate with infertility.^[45] Magnúsdóttir *et al.*^[46] observed that semen quality of obese male subjects was 3 times lower than men of normal BMI while other studies associated increase BMI with decrease sperm indices and increased sperm damage.^[47-49] Underweight also affect fertility in the male. Men who are under weight have been reported to have lower sperm concentrations compared to those with normal BMI.^[49] Therefore, body exercise of at least 3 times a week for about 1 h was necessary to keep the body healthy. It was reported that men who engage in such exercises scored higher in most sperm indices compared to those who participate in more frequent and rigorous exercise.^[50]

Cigarette smoking and alcohol

Not many studies have been done to implicate cigarette smoking in infertility. It was however reported that subjects who smoke before or during an attempt to conceive risk decreasing their fertility when compared to non-smokers.^[51] Men who smoke tend to have decrease sperm parameters since smoking could reduce the mitochondrial activity in spermatozoa and decreased fertilizing capacity.^[52] Other authors observed that about 6% of moderate and heavy smokers who were evaluated had decreased sperm indices.^[53] There is controversy regarding the effect of alcohol on fertility; whereas some studies have demonstrated an association between alcohol and infertility, it is not clear what amount of alcohol intake relates to an increased risk of infertility.^[28] Alcohol consumption has been associated with many negative effects in men including decreased libido, testicular atrophy and decreased sperm count.^[54,55]

Illicit drugs

The use of illicit drugs such as marijuana and cocaine appear to have negative effects on fertility even though

not much study has been reported. The dearth of studies on the effects of illicit drugs on human fertility has been attributed to ethical considerations, under-reporting and bias because of the characteristics of the population being studied.^[56] Marijuana is a commonly used drug, and it acts both centrally and peripherally to cause abnormal reproductive function.^[28] Cannabinoids, a major component of marijuana can bind to receptors located on reproductive tissues such as the uterus and ductus deferens. In men, it has been reported to reduce testosterone release from the Leydig cells, regulate apoptosis of Sertoli cells, decrease sperm capacitation and decrease acrosome reaction.^[57] Cocaine also acts on both peripheral and central nervous system that causes vasoconstriction and anesthetic effects. It can prevent the uptake of neurotransmitters and affects behavior as well as mood.^[58] Long-term use may lead to decrease sexual stimulation, difficulty in achieving and maintenance erection and to ejaculate.^[59] Cocaine has also been reported to affect fertility because of its ability to increase serum prolactin and decrease testosterone levels.^[60,61] It was however observed that the of cocaine depends on dosage, duration of usage and interactions with other drugs.^[62]

Changes in male reproductive system

Hormonal abnormalities

Endocrinologic disorders and infertility are common all over the world. Because several authors have suggested that infertility in Africa is due to the high prevalence of sexually transmitted diseases, we decided to evaluate the contributions of endocrine abnormalities to infertility in the male in Kano, Nigeria. Hormonal abnormalities were detected in 22% oligospermic, 40.7% in severe oligospermic and 42.7% in azospermic male subjects.^[21]

The causes of male infertility may be pretesticular, testicular and post testicular.^[63] The pretesticular and the testicular causes are mainly endocrine disorders originating from the hypothalamic-pituitary-gonadal axis that have adverse effects on spermatogenesis. Male fertility is critically dependent upon the normal hormonal environment. Therefore, appropriate evaluation and treatment of the sub-fertile men are critical in the delivery of suitable care to the infertile couple.^[21] The pattern of abnormalities observed in oligospermia was hypergonadotropic-hypogonadism and hypogonadotropic-hypergonadism, while hypogonadotropic-hypogonadism and hyperprolactinaemia were observed in severe oligospermic males. Also, primary hypogonadism, secondary hypogonadism and hyperprolactinaemia were observed in azospermic males.

Evidence in man suggests that luteinizing hormone (LH) and FSH through the action of testosterone are required for the initiation and maintenance of spermatogenesis.

Testosterone, in addition, is important in maintaining the seminiferous epithelium. This action of testosterone is mediated through androgen receptors within the Sertoli cell.^[64-67] Male hypogonadism may indicate an impaired synthesis of testosterone by the Leydig cells or insufficient spermatogenesis due to seminiferous tubular dysfunction. This is so because extensive biochemical communication occurs in the Leydig cells and the tubules. When there is a disturbance in the hormone releasing processes the whole process leading to spermatogenesis is disrupted.

The Increase in serum FSH level in azoospermia may reflect decreased testicular activity resulting in changes in normal feedback mechanism between the testes and the hypothalamic-pituitary axis.^[67] Hyperprolactinaemia was observed in two patients with oligospermia and six azoospermic males coupled with low semen volume. The mechanism by which hyperprolactinaemia leads to infertility in the male is not well understood, but it may lead to reduced gonadotropin releasing hormone (GnRH) secretion by slowing the frequency of GnRH pulses, thereby reducing LH and FSH pulsatility. It may also alter the positive feedback mechanism on the hypothalamus.^[67-69]

Hormonal profile of a group of azoospermic males was evaluated and it was observed that 40% of all azoospermic subjects had abnormal hormonal levels while 60% had normal hormonal values and 45% of the subjects had testicular pathology.^[67] It was concluded that endocrinopathies are common in azoospermia and their contribution to male infertility is great. Other studies in Nigeria recognized that azoospermia is a common finding among infertile males. The condition was present in 6.5% of males attending a general infertility clinic and 35% in those attending male infertility clinics.^[70-71]

The major causes of azoospermia such as failure of spermatogenesis and obstruction of the ductal system especially the vas deferens have been studied. It was observed that blockage of the vas deferens was not a major cause of azoospermia in Nigeria.^[72] This observation was corroborated by findings that only one subject was fructose positive of all azoospermia evaluated for seminal fructose.^[10] Seminal fluid fructose is usually done for all azoospermia to ascertain the presence or absence of vas deferens. Fructose produced in the seminal vesicles is androgen dependent and serves as a source of energy for ejaculated sperm cells. It is absent in subjects with congenital absence of vas deferentia who have no seminal vesicles and those with bilateral ejaculatory duct obstruction.

Evaluation of the infertile men requires a complete medical history, physical examination, and laboratory investigations in order to identify and treat correctable causes, otherwise,

the patients can be counseled and the partner spared of invasive procedures and potential complications associated with such procedures.^[73]

Histological examination of testicular biopsies in azoospermic condition showed that the condition was due to primary testicular defects in half of the subjects investigated. Among the abnormalities detected were spermatogenic arrest, testicular atrophy, and hypospermatogenesis. The chromosomal abnormality was observed in one subject as a genetic factor contributory to male factor infertility.^[67]

Impact of trace elements on spermatogenesis

In a study of impact of blood and seminal Zn to seminal plasma, it was reported that the ratio of distribution of Zn between blood and seminal plasma were 1:1, 1:3, 1:4 in oligospermic, normospermic and azoospermic subjects respectively.

The mean Cu level was higher in serum than seminal fluids in all groups. Also, the Zn level was significantly higher in seminal plasma than serum. The reported differences in the Zn serum-seminal plasma ratio was due to a significantly high serum Zn level in oligospermic subjects, and this is an indication of toxicity that may have contributed to poor semen quality in oligospermic subjects. The measurement of Zn is a good index of assessing prostatic secretion and function, and it may be considered useful as an adjunct to other parameters in assessing male infertility.^[74] Several trace elements have been shown to be essential for testicular development and spermatogenesis. Zinc is secreted by the prostate into seminal plasma, but Cu in seminal plasma originates from both prostate and other structures of the reproductive tract, such as the epididymis and seminal vesicles.^[75-76] Changes in trace element levels in seminal plasma may be related to the fertility capacity of spermatozoa.^[74] Trace elements are important for male reproduction as Zn is necessary for growth, sexual maturation, and nucleic acid metabolism.^[74]

Erectile dysfunction

Erectile dysfunction (ED) is a contributory factor to male infertility. The prevalence of this condition in African countries amongst patients attending primary care clinics age-standardized to the 2000 US population was 50.7% in Nigeria,^[77] 54.9% in Egypt^[78] and 54% in Morocco.^[79] In a retrospective study of 115 subjects with ED, we observed different patterns of hormonal abnormalities in 48.7% of the subjects. These include primary hypogonadism (10.4%), hypogonadotropin (26.1%) and isolated low FSH (12.2%). No endocrine disorder was observed in 51.3% of the subjects. It was concluded that endocrine disorders are common in patients with ED and, therefore, essential in the management of these patients.^[80] The findings of

reproductive hormone derangements in ED may further complicate the reproductive potentials of the patients. The risk factors for ED include age (the prevalence of ED is 5% in men in their 40s, 10% for those in their 60s, 20% in men in their 70% and 50% in those in their eighth and ninth decades), diabetes mellitus, hypertension, hyperlipidaemia, excessive caffeine consumption and smoking.^[81] Studies have shown that obesity is a risk factor ED.^[82] It was observed that 96.5% of males with metabolic syndrome presented with ED. This condition may be a consequence of the conversion of androgen to estradiol by the enzyme aromatase that is found primarily in the adipose tissue.^[83] As the amount of adipose tissue increases, the aromatase enzyme also increases and more androgen is converted to estradiol.^[83]

Genetics, anti-sperm antibodies, and aflatoxins

Studies conducted to characterize the genetic background of infertility in Nigeria have shown that 39.5% of the normospermic males have reduced CAG repeats, and 26% of the infertile subjects have moderately increased CAG repeats.^[84]

Circulating anti-sperm auto-antibodies and nonagglutinating cytotoxic antibodies were detected in 44% and 2% respectively in a group of infertile men in Calabar, Nigeria.^[85] The study concluded that the incidence of anti-sperm auto-antibodies among infertile men is high in Nigeria and may be related to the high prevalence of sexually transmitted diseases. In a similar multi-centres study of anti-sperm antibody (ASA), it was observed that out of the 202 infertile subjects, 57 (28.2%) were positive for ASA while 145 (71.8%) were negative. Twenty-five (22.7%) of the 110 male subjects were ASA positive while 32 (34.8%) of the 92 female subjects were ASA positive. The overall prevalence in males and females was 22.7% and 34.8% respectively. Seminal fluid viscosity was inversely ($P < 0.05$) co-related with ASA as ASA was present in most subjects with normal sperm viscosity.^[86]

A high concentration of aflatoxin was reported in a group of infertile Nigerian men compared to the fertile

controls. It was observed that the consumption of native diets containing this contaminant might predispose to male infertility in Nigeria.^[87] Other important factors that have been associated with male factor infertility include infections such as tuberculosis and mumps that are capable of damaging the male reproductive system directly or indirectly.^[88]

Reduced acrosin activity and calcium

We previously evaluated acrosin activity in spermatozoa of infertile Nigerian men as an indication of fertilizing capacity. It was observed that acrosin activity in infertile Nigerian men was significantly lower than the fertile men. Acrosin activity was also affected by morphological changes in the spermatozoa.^[89] Human spermatozoa require capacitation to become competent for fertilization. Acrosin is a sperm acrosomal protease that plays an important role in the process and is involved in the acrosome reaction, that is, the binding of spermatozoa to the zona pellucida and/or the penetration of spermatozoa through the zona pellucida.^[90] The mechanism of capacitation and fertilization mediated by acrosin activity was elucidated by Iannaccone.^[91] The acrosin reaction can only occur in the right calcium environment.

Emokpae and Emokpae^[92] earlier reported significant changes in calcium concentration in azoospermia and oligospermia when compared with normospermia. There were also changes in seminal plasma calcium levels of azoospermia when compared with oligospermia. The study indicated that calcium levels in the seminal fluid increased with increasing seminal cell density and decreased with decreasing sperm cells from normospermia, oligospermia to azoospermia.

MANAGEMENT OF MALE INFERTILITY

Male infertility can be managed through counseling, hormonal and drug therapy as well as surgical intervention.

Table 1: List of some studies done in the last 10 years on male infertility in Nigeria

Authors	Study design and topics	Year of publication
Emokpae and Adobo ^[33]	Original article: Association of seminal plasma with semen quality	2015
Adejumo et al. ^[86]	Original article: Study of Anti-sperm antibodies among infertile subjects	2014
Onyeka et al. ^[26]	Original article: Semen analysis of 263 men from infertility clinic	2012
Akinloye and Truter ^[15]	Review article: Review of management of infertility in Nigeria	2011
Akinloye et al. ^[74]	Original article: Impact of blood and semen plasma zinc and copper concentration on spermiogram and hormonal changes	2011
Akinloye et al. ^[84]	Original article: Androgen receptor gene CAG and GGN polymorphism	2009
Emokpae et al. ^[23]	Original article: Contributions of Bacterial infection to male infertility	2009
Ekhaise and Omoregie ^[16]	Original article: Common Bacterial isolates in infertile males	2008
Ojobo ^[18]	Review article: Management of infertility amongst Nigerian couples	2007
Emokpae et al. ^[21]	Original article: Male infertility and endocrinopathies	2007
Emokpae et al. ^[67]	Original article: Hormonal abnormalities in azoospermic men	2006
Emokpae and Uadia ^[89]	Original article: Acrosin activity in spermatozoa of infertile males	2006
Emokpae et al. ^[80]	Original article: Biochemical pattern of erectile dysfunction in Nigerians	2006
Okonofua et al. ^[7]	Original article: Case control study of risk factors for male infertility	2005
Emokpae et al. ^[22]	Original article: Semen quality and infection	2005

The prognosis depends on the duration of infertility, primary or secondary infertility, results of the seminal analysis, age and fertility status of the female partner.^[93] Depending on the life style of the subject, he could be counseled against heavy smoking, alcohol abuse, use of anabolic steroids, engaging in extreme sports, wearing of thermal underwear and occupational exposure to heat sources. The use of antioxidant supplements such as Vitamins E, A and C, zinc, selenium and antibiotics administration may help to improve semen quality. In subjects with endocrine abnormalities, the use of drugs and endocrine replacement therapies may be helpful. Surgical treatment may be carried out to correct pathological conditions that include varicocele and epididymal obstruction. Anejaculation can be treated by vibro-stimulation or electro-ejaculation techniques.^[94] However, with most infertility attributable to idiopathic oligospermia and other sperm abnormalities, interventions have failed to resolve this condition. Improvements in technologies have made assisted reproductive techniques possible.^[94] Techniques such as intra uterine insemination, *in vitro* fertilization, sperm extraction techniques, and micro assisted fertilization are now available. These procedures are beyond the reach of the ordinary man on the street, hence the urgent need for government and other donor agencies to assist in the treatment of male infertility.

CONCLUSION

Male factor infertility in Nigeria accounts for up to 50% of all cases. The major causes are poorly treated STIs and hormonal abnormalities. Effort should be made at arriving at a proper diagnosis, and adequate treatment given where causes are treatable otherwise the patients should be adequately counseled. Given that health care facilities are limited in Nigeria and that infertility is difficult and costly to manage, and resources are limited, it is imperative to focus more on prevention. Preventive measures should include sex education, maintenance of public health and hygiene, effective treatment of STIs, early treatment of abnormal physical conditions and prevention of damage to testes from trauma. Public health policy makers should make available and increase the allocation of resources aimed at improving male factor infertility. Agencies should be established to focus on prevention and management of male factor infertility. In cases where correction is not possible, assisted reproductive techniques may be suggested. This procedure as at now is beyond the reach of the average citizen. Centers where such facilities are available should be subsidized by the government to reduce the cost. In addition, adoption should be encouraged to reduce the number of children in the various orphanage homes [Table 1].

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