Original Research Paper Gynaecology

EVALUATION OF MALE FACTORS CONTRIBUTING INFERTILITY

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ABSTRACT

Introduction : Infertility is a public health problem in developing countries like India with serious social implications on affected couples and families. Though a lot has definitely been achieved in terms of understanding the process of reproduction but the etiology of male infertility is still unclear in many situations.hence we decided to do the study

Material and Methodology : 100 husband of infertile couples were selected for the study from infertility out door from Jul 2015 to Jun 2017, a thorough history and complete physical examination was recorded as per proforma with other history components. After 3-5 days of ejaculatory abstinence the semen samples were collected in a sterile plastic container by the process of masturbation from the subjects (WHO, 2010). Analysis was done according to the World Health Organization (WHO,2010) Guidelines. Scrotal USG was done to identify scrotal and testicular pathology. Serum FSH, LH and Testosterone levels were assessed.

Results : Males in the study group were aged between 20-58 years of which maximum number comes between 31-40 years age group(47%), Majority of the patients (56%) were lying in BMI group between 20 – 24.9 kg/m2,22% of the patients were in the BMI group 25-29.9kg/m2 and 2% patients were obese BMI is more than 30 kg/m2. Regarding the type of addiction majority of the male partner that is 53.57% are having habit of tobacco chewing, 22.62% males are having the habit of smoking and 23.81% were addicted to alcohol. All the conditions were statistically significant. 35% were having normospermia, 17% were having oligoasthenospermia, 14% are having azoospermia and 13 % were having asthenozoospermia. The mean FSH and LH levels were significantly increased ($P<0.05$) in infertile males with azoospermic, oligospermic, infertile males with varicocele, where as testosterone levels were less. azoospermic patients has testicular size of average 14.07ml in comparison with normospermic subjects (N=35) having average size of testicles of 20.65ml

Conclusion : Our study provides mere insight into where the greatest need is for further research into underlying etiology and treatment correction of anatomical defects, refraining from addiction with optimum time of marriage, treatment of the underlying hormonal imbalance and infection will reduce the number of cases of male infertility.

KEYWORDS :

INTRODUCTION

Childlessness may be a tragedy to the married couple and can be a cause of marital upset as well as of personal unhappiness. To have a child cements a marriage. The desire of a couple for children is usually stronger then self interest in beauty and figure and may be stronger than the claims of a carrier.[1]

Of all sexually active couples, 12-15% are infertile, as per general population survey. When broken down by gender a male component can be identified 50% of the total infertile couple either in isolation or in combination with a female factor[2].

The prevalence rate varies between and within countries. For instance in United Kingdom and the USA is estimated to be 6% and 10% respectively[3].In Nigeria and sub-Saharan countries, infertility rate could exceed 30%. The high level of infertility in Africa is due largely to reproductive tract infections which lead to abnormal semen parameters and low sperm count[3]. There is one data available from Kashmir valley in India which is 26-32%[4]. Unfortunately there are not many studies available in India, hence we have decided to conduct the above mentioned study to find out different causes of male infertility.

AIMS AND OBJECTIVES

AIMS:

- To find out different etiological factors contributing male infertility

OBJECTIVE:

Male factor infertility is a complex disorder that affects a large sector of the population; however, many of its etiologies are unknown. By elucidating the underlying causes including, it may be possible to discover the exact causes of infertility and hence to send the patients to respective departments for accurate modality of treatment, avoiding unnecessary delay.

MATERIAL AND METHODOLOGY

MATERIALS

Place of Study: Acharya VinobaBhave Rural Hospital;Sawangi; wardha.

Duration of study- JULY 2015-JUNE 2017

Study design- Diagnostic analytical study.

My study involved

a) Dept. of Obstertics and Gynaecology.
b) Dept. of Physiology and Wardha test tube baby centre for Semen Analysis.
c) Dept. of Radiology for scrotal sonography
d) Dept. of Biochemistry for FSH,LH & Testosterone assesment

And result was compared with current similar studies done in other centres and one master chart was prepared as par WHO recommended semen analysis chart.

Sample size – 100 cases were selected for the study from infertility out door, a thorough history and complete physical examination was recorded as per proforma with other history components.

Inclusion criteria – Husband of all infertile couples were recruited for semen analysis.

Exclusion criteria

All males below 21 and above 60 years of age were not considered

for the study. Men with genetic disorder, Erectile dysfunction, cardiovascular problem, HIV positive and Hepatitis (HBsAg) positive were excluded from the study.

METHODOLOGY

After taking written consent from both the partners the following methods were adopted. History was taken and noted regarding – Duration of Marriage, co-habitous, history of any childhood infections like mumps, STD, tuberculosis, viral infection. Sexual history like potency, libido, lubricant used, time and frequency of intercourse. Personal history of any systemic disease like diabetes, cirrhosis, hypertension were noted. Exposure to heat, alcohol, cocaine, cannabis abuse, tobacco use and radiation exposure was noted.

History of previous surgery like orchidopexy, herniorraphy, orchidectomy pelvic and perineal surgery, bladder neck surgery were noted.

Physical examination

A general physical examination is an integral part of the evaluation of male infertility. In addition to the history with general physical examination, particular focus was given to the genitalia including 1) examination of the penis; including the location of the urethral meatus; 2) palpation of the testes and measurement of their sizes; 3) presence and consistency of both the vasa and epididymis; 4) presence of a varicocele; 5) secondary sexual characteristics including body habitus, hair distribution and breast development;and 6) digital rectal exam. The diagnosis of congenital bilateral absence of the vasadeferentia (CBAVD) was established by physical examination.

Semen Collection and Analysis

After 3-5 days of ejaculatory abstinence the semen samples were collected in a sterile plastic container by the process of masturbation from the subjects (WHO, 2010).

Semen samples were collected in the laboratory room in a clean, dry, biologically inert container. In case of oligozoospermic or azoospermic patients, three semen samples were collected 3 times on different days with three days abstinence and thorough examination was carried out. The collected samples were allowed to liquefy at 37°C for 30 minutes and analyzed within one hour after collection.

Specialized clinical tests

1) Ultrasonography

Ultrasonography was done to find out abnormalities of the male genital tract that may adversely effect fertility. Scrotal USG was done to identify scrotal and testicular pathology, to take the measurement of the testis and pathology like absent vas deferens, epididymal induration and testicular masses.

Quantification of leukocytes in semen

The presence of leucocyte concentration in semen were assessed by myeloperoxidase staining technique. Peroxidase positive WBCs staining dark brown were counted in all 100 squares of grid in Makler's chamber under bright field objective (magnification, 20x). The results were recorded. WBC Count > 10^6/ml were considered as leucocytospermia.

Endocrine evaluation

Endocrine evaluation was performed. Serum FSH, LH and Testosterone levels were assessed.

RESULTS

Males in the study group were aged between 20-58 years of which maximum number comes between 31-40 years age group(47%), followed by 21- 30 years age group (41%) and 12 % were above 41 years age. Majority of the patients (56%) were lying in BMI group between 20 – 24.9 kg/m^2,22% of the patients were in the BMI group 25-29.9kg/m^2 and 2% patients were obese BMI is more than 30 kg/m^2. Majority of the patients in the present study were coming under the category of primary infertility (63%) & secondary infertility 33%. 54% of the infertile male partner were having duration of infertility of 4 – 7 years, followed by 20% having duration between 8-11 years and 14% were having more than 11 years . Regarding the type of addiction majority of the male partner that is 53.57% are having habit of tobacco chewing, 22.62% males are having the habit of smoking and 23.81% were addicted to alcohol. All the conditions were statistically significant. While observing different infertility conditions among the infertile male 35% were having normospermia, 17% were having oligoasthenospermia, 14% are having azoospermia and 13 % were having asthenozoospermia, 8% were having oligozoospermia,8% teratospermia and 5% oligoasthenoteratospermia. We found out the following anatomical defects, history of orchidopexy 2%, varicocele 2 %, history of mumps 1%, hydrocele 1%, history of perineal injury 1% and history of hernioplasty 2%. So total 9% of cases suffers from anatomical abnormality as per history and clinical examination.

Table 1: Serum FSH, LH and testosterone levels in different subgroups of infertile males

Sr. No.	Group	Number of Subjects	Hormone levels mean ± SD		
			FSH (mlu/ml) (Range)	LH (mlu/ml) (Range)	Testosterone (ng/ml) (Range)
1	Normospermia	35	9.44±1.84 (3.89-16.62)	7.96±1.49 (5.44-13.48)	5.48±0.50 (4.17-6.16)
2	Azoospermia	14	15.04±4.09 (3.65-20.61)	13.37±4.34 (8.43-25.48)	4.51±0.58 (3.45-5.41)
3	Oligozoospermia	8	13.81±2.55 (11.03-18.60)	12.42±4.26 (8.87-21.50)	4.38±0.86 (2.86-5.16)
4	Asthenozoospermia	13	14.35±3.02 (9.43-19.75)	10.29±1.94 (6.67-13.07)	4.87±0.71 (3.68-6.43)
5	Oligoasthenospermia	17	14.51±2.24 (9.49-18.73)	11.39±1.92 (7.69-14.47)	4.47±0.55 (3.09-5.16)
6	Terratospermia	8	13.31±1.64 (10.66-16.23)	10.74±1.71 (8.45-13.85)	4.88±0.65 (3.59-5.81)
7	Oligoasthenoteratospermia	5	15.36±2.60 (12.83-18.60)	11.06±1.59 (8.63-12.68)	4.61±0.36 (4.02-5.01)
	Total	100	12.68±3.48 (3.65-20.61)	10.34±3.12 (5.44-25.48)	4.91±0.72 (2.86-6.43)

Table 1 – regarding hormonal abnormalities in different subgroups of infertile subjects 35 normospermic males are having FSH level 9.44 ± 1.84 mlu/ml, LH level 7.96 ± 1.49 mlu/ml and testosterone level 5.48 ± 0.50 ng/ml.

14 azoospermic subjects are having raised FSH level 15.04 ± 4.09 mlu/ml, LH level 13.37 ± 4.34 mlu/ml and low testosterone level 4.38 ± 0.58 ng/ml.

8 number oligospermic subjects are having raised FSH level as 13.81 ± 2.55 mlu/ml, raised LH level 14.4 ± 4.26 mlu/ml and low testosterone level 4.38 ± 0.58 ng/ml.

13 number of asthenozoospermic individuals are having raised FSH level 14.35 ± 3.02 mlu/ml, raised LH level 10.29 ± 1.94 mlu/ml and low testosterone level 4.87 ± 0.71 ng/ml.

17 number of oligoasthenospermic patients are having raised FSH level 14.51 ± 2.24 mlu/ml, raised LH level 11.39 ± 1.92 mlu/ml and decreased testosterone 4.47 ± 0.55 ng/ml.

Teratospermic subjects of number 8 are having raised FSH 13.31 ± 1.64 mlu/ml, raised LH 10.74 ± 1.71 mlu/ml, and decreased testosterone 4.88 ± 0.65 ng/ml.

Oligoasthenoteratospermia 5 cases having raised FSH 15.36 ± 2.60 mIu/ml, raised LH 11.06 ± 1.59 mIu/ml and decreased testosterone 4.61 ± 0.36 ng/ml.

Table 2: Testicular volume of the subject with different type of seminogram

Sr. No.	Group	Number of Subjects	Testicular volume mean ± SD			
			Right Testis (Range)	p-value	Left Testis (Range)	p-value
1	Normospermia	35	20.65±0.84 (19-22)	-	20.61±0.89 (19-22)	-
2	Azoospermia	14	14.07±1.36 (12.50-18)	0.0001,S	13.20±0.86 (12-15.80)	0.0001,S
3	Oligozoospermia	8	15.68±1.98 (12-18)	0.0001,S	15.68±1.41 (13-17)	0.0001,S
4	Asthenozoospermia	13	17.59±0.86 (16-19)	0.0001,S	16.76±1.07 (15-18.50)	0.0001,S
5	Oligoasthenospermia	17	17.17±1.07 (15-19)	0.0001,S	16.84±0.79 (15.50-18.60)	0.0001,S
6	Terratospermia	8	17.18±0.92 (16-18.50)	0.0001,S	16.75±1.79 (14-20)	0.0001,S
7	Oligoasthenoteratospermia	5	16.60±1.98 (13.50-18.50)	0.0001,S	16.36±0.92 (15-17)	0.0001,S
	Total		17.86±2.57 (12-22)	0.0001,S	17.52±2.75 (12-22)	0.0001,S

Graph 2: Testicular volume of the subject with different type of seminogram

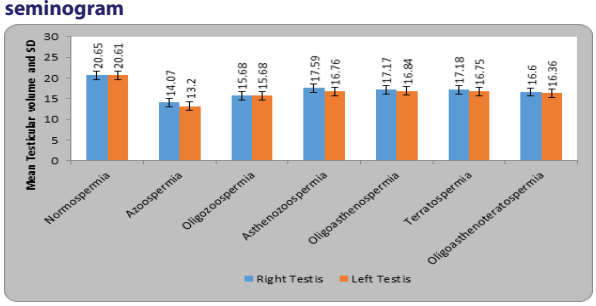

Table 2 – 35 normospermic subjects are having testicular right sided volume having average size of 20.65 ± 0.84 ml and left testis volume 20.61 ± 0.89ml where as 14 number of azoospermic subjects are having average right sided testis volume 14.07 ± 1.36ml and left 13.20 ± 0.86 ml. oligospermic 8 subject having volume of testicals right 15.68 ± 1.98ml, left 15.68 ± 1.41ml.

Asthenospermic 13 cases having low right testicals volume of 17.59 ± 0.86ml and left testis volume 16.79 ± 1.07ml, in oligoasthenospermic 17 subjects having testicular volume right side 17.17 ± 1.07ml and low left testis volume 16.84 ± 0.79ml.

8 number of teratospermic subjects having low testicular volume right side 17.18 ± 0.92ml and left 16.76 ± 1.79ml.

5 number of oligoasthenoteratospermic subjects are having low testicular volume i.e. right 16.60 ± 1.98ml and low left sided testicular volume 16.36 ± 0.925ml.

Table 3: Types of semenogram with respect to Leucocytospermia

Types of Semen abnormality	No. of cases of Leucocytospermia	% of cases of Leucocytospermia
Teratospermia	3	3
Asthenozoospermia	2	2
Oligospermia	1	1
Total	6	6

Table 3 shows total 6 patients having Leucocytospermia out of which 3 from Teratospermia group, 2 from Asthenozoospermia group and 1 from Oligospermia.

DISCUSSION

Male infertility is a global population health concern. There is an estimated 48.5 million couples, suffer from infertility world wide. Over all, by examining the available literature and consolidating the information the global rates of male infertility ranges from 2.5 to 12% **(Ashok Agrawal).**[2] Because 80% of couples are able to achieve the pregnancy within the 1st year of attempting, a couple should only be diagnosed as infertile after 1 year of regular sexual activity without using any contraceptive method. For healthy young couples the possibility of achieving pregnancy per reproductive cycle is approximately 20-25%. The cumulative probabilities of conception are 60% within 1st six month, 84% within 1st year and 92% within the 2nd year of fertility focussed sexual activity **(Kamel 2010).**[5] Andrologist is responsible for diagnosing, counselling and treating the underline cause. When ever possible. When there is no specific he or she is still responsible for referring the patients to specialised ART centre.

Hasan and Killick (2003)[6] stated that increased male age is associated with a significant declined in fertility, which is independent of the women's age, coital frequency and life style effect. In addition, paternity at older ages may have significant effects on the viability and genetic health of human pregnancies and offspring's. in our study maximum number of infertility cases belongs to 31 to 40 years age group, reflecting increase age diminishes sperm count and this is in agreement with previous studies.

Increased BMI is a major health issue and the relationship between obesity and male infertility has been described recently in many reports, also men with high BMI typically are found to have an abnormal semen analysis as well. **(Jensen et al 2004)**[7] reported a higher prevalence of oligozoospermia in over weight and obese man compared with normal BMI, however they did not find any relationship between increase in male BMI and percentage of motile sperm. **(Kort et al 2006)**[8] found that BMI correlated negatively with total number of normal spermatozoa. In a recent study in India the negative correlation was found between male BMI and sperm parameters like sperm count and motility and stated that obesity may lead to male infertility by lipid peroxidation **(Najafi et al 2011)**[9]. Semen abnormalities are associated with increased body weight as the percentage men with abnormal sperm volume, concentration and total sperm count increased with increase in size **(Eisen Berg et al 2014)**[10]. In contrast are recent meta analysis pooling 5 studies has found no relationship between BMI and semen parameters **(McDonald et al 2010)**[11]**.** Other factor associated with obesity such as central leptin resistance and the increase in some adipokines **(Fischer – Posovszky et al 2007**[12]**, Gautier et al 2013**[13]**)** may also participate in the pathophysiology of male obesity associated secondary hypogonadism (MOSH), by inhibiting gonadotropin pulse and or secretions.[13]

Several studies have reported reduction in testosterone with obesity. Many studies lagged information on frequency of sexual intercourse having obesity related changes in sexual function could not be distinguished from obesity related on fertility **(Sallmen et al 2006**[14]**),** because obesity has been associated with sexual and erectile dysfunctional **(Esposito 2005**[15]**),** therefore reduces intercourse could be a mediating factor by which obesity producess infertility. In our study maximum number that is 56% of the infertile males are coming under BMI of 20 to 24.9kg/m^2 which authenticates the hormonal factor and reduction of testosterone synthesis.

Abubakar A Panti[16] et al in 2014 observed the prevalence of infertility in Nijeria was 15.7% out of which 32.8% had primary and 67.2% had secondary infertility, on contrary to the fact **Mohammad Ayaz Khan**[17] et al from Lahore in 2012 observed 83.2% patients have

primary infertility and 16.8% were suffering from secondary infertility. In our study we have found out 63% of the infertile males suffer from primary infertility and 37% suffers from secondary infertility which is in accordance with Lahore study by MD khan et al. In epidemiological study of male infertility by **Dr. S. Samal**[18] et al in May 2012 reported that 61.98% of the infertile males are having normospermia and rest are having seminal abnormalities. In a study by **P Choudhary**[19] et al in 2015, they found out of 100 cases of semen analysis, 63% had normospermic and rest were having abnormal seminogram. In our study we found out of 100 cases of semen analysis 35% had normospermia.

In a study by **Bodal V K**[20] et al in January 2014 showed that couples with duration of marriage between 3 to 6 years were 45% followed by couple with duration 1 to 3 years in 44% and 6 to 9 years is 10% and least number of those whose duration of marriage is 9 to 12 years is 1%. In another study by **Atul Jain**[21] et al in 2016, observed 66% had duration of marriage was below 5 years, 20% between 5 to 10 years and 14% with more than 10 years of marriage. In our study majority of the subjects were that is 54% are coming under duration of infertility for 4 to 7 years, 20% of the affected males are coming in the duration of infertility between 8 to 11 years, 14% of the patients were in the category of above 11 years of duration of infertility and 12% affected males are coming under the category of 1 to 3 years of duration of infertility which corroborate with contemporary studies. In a study by **Renu Jain**[22] et al in 2015, reveals a significant decrease in sperm motility in smoking individuals. However **Mery**[23] et al similarly observed a decrease in type I and II motility but increase in type IV motility in smoking candidates, those finding were consistant with other studies. The concentration of sperms was found to be significantly more in non-smoking individuals. **Chia**[24] **et al and Merino**[25] **et al** also observed decrease in concentration of sperms in smoker individuals. In a study by **Dushayant Singh Gour**[26] et al in 2010 found that cigarette smoking appears to contribute significantly towards impairment of sperm motility. Also, asthenozoospermia appears to be the earliest defect of sperm quality as seen by its dominance over other defects amongst light smokers. Alcohol abuse in men has been reported to cause impaired testosterone production and atrophy of testis which can results in impotency, infertility and reduced male secondary characteristics. In a study by **S. Samal**[18] et al in 2012 had showed that in no addiction group 82.96% were normozoospermia while 17.06% had abnormal semen analysis report which was highly significant. It was found that addictions like smoking, alcohol and combination of these had a detrimental effect on spermatogenesis. In our study in no addiction group 17% affected males were found, where as 83% of affected males were found to be in the addiction group.

In a study by **Atul Jain**[21] et al 28% of their study subject had normal morphology and 72% had abnormal morphology. In another study by **Ugboaja**[27] et al 2010 studied the pattern of seminal fluid abnormality in male partners of infertile couples in south eastern Nijeria, over a period of 12 months and it was found, out of 348 semen sample reports evaluated 238 have semen fluid abnormalities. Asthenozoospermia 16.7% was the single main abnormality followed by oligoasthenozoospermia.

Salgado[28] et al 2003, conducted a study on 571 infertile couples and found that asthenozoospermia was present in 8.89% of cases. In a study by **Bodal V.K.**[20] et al 2014 the male factors was responsible in 43% cases as a cause of infertility and asthenozoospermia (17%) was the most common type of semen defect present in these infertile males, followed by oligoasthenozoospermia.

In a study by **Mohammad Ayaz Khan**[17] et al 2012 they analysed semen in 416 patients. 49.8% of them where having normal count, 22.4% had azoospermia, 18.5% had oligozoospermia. Examination of sperm morphology revealed that 79% had normal morphology and 21.4% had asthenozoospermia.

In our study out of 100 infertile subjects 35% have normospermia, 14% had azoospermia, 17% had oligoasthenozoospermia, 13% having asthenozoospermia, 5% oligoasthenoteratospermia and 8% teratospermia which corroborates with the studies mentioned.

Marimuthu[29] et al 2003, conducted semen analysis of subjects attended the fertility clinics for the last 11 years observed that the average age of men attending the infertility clinic was 31.2 years. The reason of alteration of seminal index in that age group was that increase age of male, there occurs physiological alteration in various semen parameters. More over majority of our population is still uneducated, they always report late to infertility clinics and in our society males are rarely considered at problem, so they rarely gate tested for infertility and seminal examination.

Priya Narayanan[30] et al in February 2017 quoted that the production of the reproductive hormone, sexual function and semen production are effected by increasing paternal age. These affectes the fertility, pregnancy outcome and some birth defects and diseases of the offspring are all related to paternal age. The impact of male age on histopathological aspect in the aging testis leads not only to reduce the number of sertoli cells, leydig cells and germ cells but also to other changes like thickening of the vessel membrane of the seminiferous tubules, parallel to a reduction in the seminiferous epithelium and defective vascularization of testicular parenchyma. Semen volume and seminal fructose concentration decreases with age, possibly due to seminal vesicle insufficiency. In our study majority of partner with semen abnormality were in the age group of 31-40 years of age, 2nd highest in 41% in the age group of 21-30 years age findings are similar to above studies.

In a study by **Berniza Calderon**[31] et al in 2016 coated that the pathophysiology of the relationship between abnormal sperm production and adiposity is uncertain and complex. Alteration in the HPO axis can lead to relative decline in gonadotropin level **(Hofstra et al**[32] **2008 an Michalakis et al**[33] **2013)**. However in author's study they could not find low level of gonadotropin in patients with subnormal semen parameters, but found that serum estradiol inversely correlated with the number of spermatozoa. The author found that alteration in semen parameters were associated with increased BMI and excess body weight in agreement with a recent study (**Eisenberg et al**[10] **2014).** It is possible that other factors may play a role in the pathophysiology of the semen alteration beyond that of gonadotropin in obese patients including an increase in testicular temperature and other life style, nutritional or environmental factors among others **(Sharpe and Franks**[34] **2002).**

In one study by **Nicole O Palmer**[35] et al from Australia in 2012, commented that one side effect of obesity may potentially contribute to alter sperm production / paramenters which raises gonadal heat resulting from increased scrotal adiposity. The process of spermatogenesis is highly sensitive to heat, with optimal temperature ranging from 34-35°c by humans. Increased testicular heat is associated with reduced sperm motility and increase sperm DNA damage and increases of sperm oxidative stress. Changes to testicular temperature can occur via a number of mechanism like physical disorder (Varicocele), increase scrotal adiposity or environmental disturbance like prolong bike ride are associated with reduced sperm function and subfertility. There is a single study which investigator the surgical removal of scrotal fat reported an improvement in sperm parameters.[35]

In our study 22% infertile male partners were belong to category of BMI of 25-29.9kg/m^2, where as majority of the patients that is 56% belong to 20-24.9 kg/m^{2}BMI group. Only 2 obese male were found to have abnormal seminogram which corroborates the above mentioned studies.

One study by **Priya Narayanan**[30] et al in 2017 observed age wise analysis of seminal fluid abnormality shows an overall similar picture of abnormalities according to various age group, changes in

sperm concentration with increase in longer duration of infertility shows a 3.3% decline with age while other data reports no change in sperm concentration up to age of 50 years.

Similarly in our study 5 number of patients of infertile male have normospermia even after 11 years of infertility, 8% affected male develop asthenozoospermia within 4 to 7 years of infertility and 11% develop oligoasthenospermia within the same period of infertility.

Dushyant Singh Gaur et al[26] quoted that alcohol has been shown to have a deleterious effect at all levels of male reproductive system. Alcohol interferes in the feedback mechanism of HPO axis, resulting in impairment and production of adequate quantity of FSH and LH leading to deterioration of sertoli cells. Alcohol affect the leydig cell and reduces blood level of testosterone by reducing its production and increase in metabolic clearance[37]. Thus alcohol induces reduction in level of testosterone, LH and FSH. Not only hamper their morphological development and maturation of spermatozoa (producing significant teratosperima) it also slows down the sperm production by testicular germ cells (oligozoospermia), specially in heavy alcoholics[38]. Other studies have reported partial to complete spermatogenic arrest among moderate to heavy alcohol consumers, leading to sertoli cell only syndrome in advance cases indicating severe testicular damage. Progressive damage to testis and reduction of sex hormone leads to loss of secondary sexual characteristics and development of impotency and infertility[66]. In our study 14% of patients suffer from some form of sperm abnormalities with moderate to severe type of alcohol abuses. 13% of smokers (moderate to severe) suffer from some or other form of sperm abnormalities. 30.3% chronic tobacco chewer suffer from some form of semen abnormalities.

Heavy exposure to alcohol and or smoking ultimately deteriorate all aspect of semen quality. Synergistic effect of alcohol intake and smoking has been reported to be very detrimental to reproductive health of an individual[39].

There is history of undescended testis in two patients in whom orchidopexy operation was done at the age of 8-10 years

Varicocele is vascular abnormality of scrotum that is defined as pathological dilatation of venous pampiniform plexus, resulting in absence or incompetence of the valve of the internal spermatic vein[40].

Previously the incidence of mumps were more, but now at the advent of vaccination of mumps reduces the incidence of mumps, which in turn affects the spermatogenesis causing azoospermia. In our study we found two cases of orchidopexy operation, two cases of varicocele, one mumps, one hydrocele, two cases of hernioplasty operation and one case having history of perineal injury.

In a study by **S. Ramesh Babu** et al[41] in 2004 from Hyderabad studied 96 infertile male and FSH, LH and testosterone levels were evaluated. The mean FSH and LH levels were significantly increased ($P<0.05$) in infertile males with azoospermic, oligospermic, infertile males with varicocele, where as testosterone levels were less in all the above mentioned clinical conditions which is similar with our study.

In one study by **OEC et al**[42] in the year 2012 at university of Orlu, commented azoospermia in the presence of high FSH and LH might indicate primary testicular failure which can be confirmed by testicular biopsy. Azoospermia with normal serum FSH and LH indicates normal spermatogenesis with blockage in the transport system.

In a review article by **Jonathon Jarrow et al**[43] in 2011 recommended an initial endocrine evaluation should include at least serum testosterone and FSH when there is an abnormal sperm count, impaired sexual function and other clinical findings suggestive of endocrinopathies.

In a review article by **Patrick O' Uadia et al**[44] in 2015 from Nijeria commented that LH and FSH to the action of testosterone are required for initiation 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. When there is a disturbance in the hormone releasing process, the whole process leading to spermatogenesis is disturbed. The increase in serum FSH level in azoospermia may reflect decrease testicular activity resulting in changes in normal feedback mechanism between the testis and HPO axis.

In a study by **Sharath Kumar** et al[45] in 2013 opine that correlation exist between testicular function and testicular ultrasonic volume, testicular quality expressed in sperm count is reflected in ultrasonic texture and in reduced testicular volume by ultrasound. It is predicted that ultrasound will occupy a major place in clinical evaluation of testicular function in future. The ultrasonic testicular volume was positively correlated with sperm count which is in accordance with our present study.

Another study by **Vinayaka U S et al**[46] in 2014 found out small testicular size is associated with decreased sperm count and as the testicular size increases the sperm count also increases. Ultrasound scan of scrotum is a good modality of investigation in evaluation of male infertility while assessing the testicular size by indirectly assessing the possible sperm count, and can be utilized in the initial assessment of male infertility.

Sanchez G.A. et al[47] in 2004 concluded in their study that testicular ultrasound should be performed in every patients of unexplained infertility and abnormal semen analysis. It allows diagnosis in depth pathological conditions than physical examination. Beside a rapid varicocele screening colour Doppler ultrasound allows us to evaluate its haemodynamic spectral display. It also provide us an exact measure of testicular volume, allows to detect the presence of dystrophic changes in testicles, as well as anomalies of the epididymis and vas deferens. They have studied 439 infertile males between 1998 to 2004.

In our study azoospermic patients has testicular size of average 14.07ml in comparison with normospermic subjects (N=35) having average size of testicles of 20.65ml which is in agreement with above mention studies.

Aduloju et al[48] in 2016 from Nijeria in their study on "Pattern of semen parameters and factors associated with infertility in male partners of infertile couples : they opined that penile contamination of the semen during collection despite the aseptic technique or it may also be due to male genital infection which is an important etiological factor in male infertility which may lead to distortion of the process of spermatogenesis, impairment of sperm function and obstruction of the seminal tract.

Dipali K. Chatur et al[49] in 2013 from India in their study on effect of leucocytospermia on seminal parameters of human male infertile subjects concluded that leucocytospermia (leucocyte count > 10^6/ml) significantly increased the rate of spermatozoa lipid peroxidation to the level which adversely affected the seminogram parameters (sperm concentration,% motility and % of normal morphology) leading to male infertility.

In our study we have found 6% cases with leucocytospermia. Of which 3% cases were having teratospermia, 2% cases were asthenozoospermia, and 1 sample was having oligospermia. Semen culture was performed of which two samples were having growth positive result. 1 having *Staphylococcus aureus* growth was from teratospermia, another sample showed growth of *Trichomonous vaginalis* which was from asthenozoospermia. Which are in

accordance with the above mentioned studies.

CONCLUSION

The male infertility is a global health problem. Which has not been researched or studied conclusively to understand its magnitude and prevalence. Our study provides mere insight into where the greatest need is for further research into underlying etiology and treatment correction of anatomical defects, refraining from addiction with optimum time of marriage, treatment of the underlying hormonal imbalance and infection will reduce the number of cases of male infertility.

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