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ORIGINAL ARTICLE

Epidemiology of Male Infertility at a Tertiary Hospital in Eastern India

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ABSTRACT

Introduction: The prevalence of infertility in the general population is 15 to 20%. Of this, the male factor is responsible for 20 to 40%. In Indian couples seeking treatment, the male factor is the cause in approximately 23% of the cases. In a World Health Organization multicenter study, 45% of infertile men were found to have either oligozoospermia or azoospermia. A study from a tertiary care hospital in India reported 58% azoospermia and 24% oligozoospermia in infertile men.

Aims and objectives: To analyze the epidemiology of male infertility.

Results: In this study of 100 cases of male infertility, 64% of the patients are in the age group 25 to 35 years, 31% of the patients are in the age group of >35 to 45 years, 4% of the patients are in the age group of more than 45 years, and 1% of the patients are in the age group of <25 years. Of the total patients, 34% (n = 37) are business people, 5% (n = 5) are clerks, 2% (n = 2) are contractors, 13% (n = 13) are drivers, 2% (n = 2) are factory workers, 6% (n = 6) are farmers, 5% (n = 5)are government workers, 3% (n = 3) each are hotel workers and jute mill workers, and 2% (n = 2) each are laborers, painters, and tea stall workers.

Conclusion: Male infertility is multifactorial: Age, occupation, and habits have a significant impact on the seminal parameters. Modifiable behaviors like cessation of smoking and alcohol are cost-effective in normalizing the semen parameters and thereby restoring fertility.

Keywords: Eastern India, Epidemiology, Male infertility.

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INTRODUCTION

The prevalence of infertility in the general population is 15 to 20%. Of this, the male factor is responsible for 20 to 40%.[1] In Indian couples seeking treatment, the male factor is the cause in approximately 23% of the cases.[2] In a World Health Organization multicenter study, 45% of infertile men were found to have either oligozoospermia or azoospermia.[3] A study from a tertiary care hospital in India reported 58% azoospermia and 24% oligozoospermia in infertile men.[4] Worldwide, 580 million experience infertility at some point of time in their life; of these, 372 million reside in low- and middle-income countries.[5.] According to a national survey of family growth conducted in 1995 by the Centers for Disease Control and the National Institutes of Health, male infertility is the causative factor in approximately 40% of the 2.1 million infertile young married couples in the United States. Africa has the burden of male infertility as high as 32%. A study in Mumbai concluded that tobacco chewing is strongly associated with a decrease in sperm quality and to a lesser extent with oligoasthenozoospermia or azoospermia.[6] The All India Institute of Medical Sciences, New Delhi, has reported a case of AZF microdeletion in cryptorchidism.[7] The study analyzes the epidemiology of male infertility at a tertiary care hospital in eastern India.

MATERIALS AND METHODS

This study was conducted in the infertility clinic (from August 1, 2009, to July 31, 2010) of the Department of Obstetrics and Gynecology at the Institute of Postgraduate Medical Education and Research, and SSKM Hospital, Kolkata. Hundred male patients attending infertility clinic, outpatients for treatment of infertility, and those who had abnormal semen analysis were evaluated with a questionnaire. The results were tabulated and analyzed.

RESULTS

In this study, 51.8% (n = 14) of the azoospermics have been infertile for 5 to 10 years, 33.3% (n = 9) for less than 5 years, and 14.8% (n = 4) for more than 10 years

In this study, 34% (n = 37) of the patients are business people, 5% (n = 5) are clerks; 2% (n = 2) are contractors; 13% (n = 13) are drivers; 2% (n = 2) are factory workers; 6% (n = 6) are farmers; 5% (n = 5) are government workers; 3% (n = 3)

each are hotel workers and jute mill workers; 2% (n = 2) each are laborers, painters, and tea stall workers; 5% (n = 5) are teachers; 6% (n = 6) are welders; and 10% (n = 10) are in miscellaneous occupations.

In this study, 29.4% businessmen had oligoasthenoteratospermia (OAT), 26.4% had oligoasthenospermia (OA), and 14.7% had azoospermia; 100% of contractors, 80% of the clerks, 23% of drivers, and 50% of factory workers had azoospermia; 50% of farmers had OAT; 16.6% of welders had OAT; 60% teachers had OA and 40% had azoospermia.

The table shows the distribution of the seminogram. In this study, OAT was noted in 32% (n = 32) of cases, azo in 27% (n = 27), OA in 26% (n = 26), and oligospermia in 8% (n = 8) of patients and normal results were noted in 7% (n = 7).

DISCUSSION

Table 1 shows the distribution of patients according to the age group. In this study of 100 cases of male infertility, 64% of the patients are in the age group 25 to 35 years, 31% of the patients are in the age group > 35 to 45 years, 4% of the patients are in the age group more than 45 years, and 1% of the patients are in the age group < 25 years.

The mean age of the patients is 34.89 years, the minimum age is 22 years, and the maximum age is 52 years. In one study, < 25 years age group comprised 1.7%, 26 to 35 76%, and above 36 years 22%.[8.] In another study, < 25 years comprised 7.3%, 25 to 34 years 66%, and > 35 years 26.7%.[8] In the present study, 25 to 35 years patients comprise 64% and > 35 years 31%.

In keeping with the decline in Leyden cell numbers, serum testosterone and free testosterone levels decrease by 0.4 and 1.2% per year respectively after the age of 50 years. Reduced mitochondrial steroid supply and reduced perfusion secondary to arteriosclerosis may contribute to this decline. Changes in the seminal characteristics involve reductions in seminal volume and sperm cell motility that may be secondary to age-related changes in the function of the epididymis, the prostate, and the seminal vesicles. Many studies have evaluated changes in the spermiogram with respect to age, but the selection of subjects, the age groups, and the methods of analysis is heterogeneous and the results are conflicting. The methodologically superior studies suggest that the semen volume, the percentage of motile sperm cells, and the percentage of the sperm cells with normal morphology decline with age.

Table 2 shows the distribution of the patients according to the duration of infertility. In this study, 58% of patients had infertility for more than 5 years but less than 10 years, 26% patients had infertility for less than 5 years, and 16% of patients had infertility for more than 10 years.

The mean duration of infertility was 6.95 years, the minimum duration of infertility was 2 years, and the maximum duration was 16 years.

In one study, < 1.5 years infertility comprised 8.4%, 1.6 to 2 years 5.7%, 2.1 to 4 years 34.6%, 4.1 to 8 years 34.6%, and more than 8 years 16.7%.[9] In the Sudan study, < 1.5 years comprised 17.2%, 1.6 to 4.0 years 43.3%, and > 4 years 28.9%.[9]

Table 3 shows the distribution of the patients' socioeconomic status. In this study, 53% of patients had income of > ₹5,000 but < ₹10,000, 28% had income of more than ₹10,000, and 19% had income of ₹2,000 to ₹5,000.

Table 4 and Graph 1 shows the distribution of the seminal parameters in smokers and nonsmokers. In this study, 3.5% of smokers had normal seminogram, 13.15% of nonsmokers had normal seminogram. In this study, the incidence of all seminal abnormalities is higher in smokers except for azoospermia, which was higher in nonsmokers. Among smokers, 3.5% had normal seminograms, whereas 13.15% of seminograms were normal in nonsmokers.

Table 1: Distribution of patients according to age

Age in years	*n = 100*	*%*
< 25	1	1
25–35	64	64
> 35–45	31	31
> 45–55	4	4

Table 2: Distribution of the patients according to duration of infertility

Sl. no.	*Duration of infertility in years*	*n = 100*	*%*
1	< 5	26	26
2	> 5–10	58	58
3	> 10	16	16

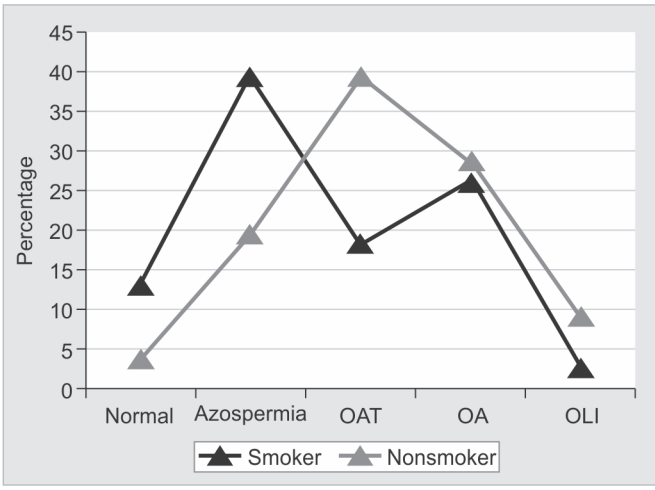
Table 3: Distribution of the patients' socioeconomic status

Sl. no.	*Monthly income (Rs)*	*n = 100*	*%*
1	< 2,000	0	0
2	> 2,000–5,000	19	19
3	> 5,000–1,0000	53	53
4	> 10,000	28	28

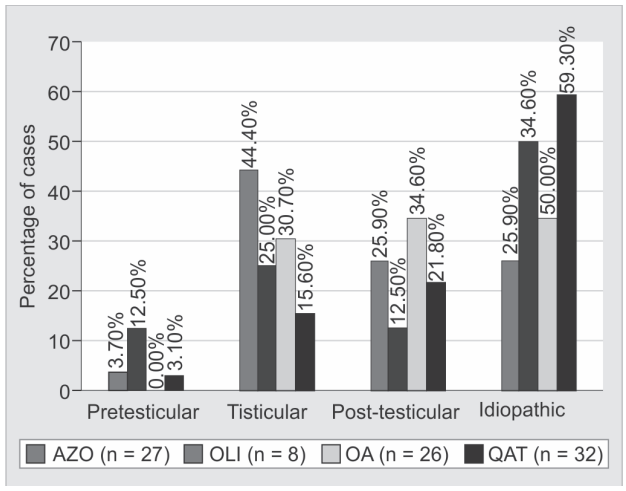
Table 4: Distribution of the seminal parameters in smokers and nonsmokers

	Nonsmoker (n = 38)	*%*	*Smoker (n = 56)*	*%*
Normal	5	13.15	2	3.57
Azoospermia	15	39.47	11	19.64
OAT	7	18.42	22	39.28
OA	10	26.31	16	28.57
OLI	1	2.63	5	8.92

OAT: Oligoasthenoteratospermia; OA: Oligoasthenospermia; OLI: Oligospermia



Graph 1: The seminal abnormalities in smokers and nonsmokers



Graph 2: The distribution of seminal abnormalities in pretesticular, testicular, posttesticular, and idiopathic groups

In one study, 63.3% of infertile men were smokers. Crude odds ratio was 2.82.[10.] Cigarette smoke contains harmful substances such as alkaloids, nitrosamines, nicotine, cotinine, and hydroxycotinine, and many of these substances are considered to generate these free radicals.[10,11] Several studies have reported that cigarette smoking is associated with reduced sperm quality (count and abnormal morphology).[12,13] Smoking also affects sperm deoxyribonucleic acid as evidenced by the increased level of 8-oxo-deoxyguanosine in spermatozoa.

In addition, infertile smokers showed higher levels of seminal Oligospermia (OS) than infertile nonsmokers.[14] Thus, the decreased sperm quality in smokers could be due to oxidative stress. Since, OS has an adverse effect on fertility potential, it is recommended that physicians advise infertile smokers to quit smoking. The effect in smokers could also be due to leukocytes in the semen, as the number of leukocytes was significantly higher in smokers.

Table 5 and Graph 2 shows the distributions of the seminal abnormalities in various groups. In this study, the majority of patients had OAT (32%). The second most common abnormality is azoospermia (27%). Oligoasthenospermia is seen in 26% and least common abnormality is oligospermia, in 8% cases. Majority of azoospermics are in the testicular groups (44.4%). A total of 3.7% azoospermics are seen in the pretesticular group. Equal number of azoospermics is seen in posttesticular and testicular groups. However, 50% of oligospermics are in the idiopathic group, 25% are in the testicular group, 12.5% are equally in pretesticular and posttesticular groups. Of the OA cases, 30.7% are in the testicular group, 34.6% in the posttesticular and idiopathic group no. cases of OA are found in the pre testicular group.

Majority of the OAT is seen in the pretesticular group (59.3% cases) and the least OAT in the idiopathic group. The posttesticular group has 21.8% OAT, and the testicular group 15.6%. In one study, idiopathic azoospermia was seen in 0.5% cases.[15] In a study at Duhok, Iraq, idiopathic azoospermia was seen in 13% of the cases.[16] In the present study, it is seen in 25.9% cases.

In a study at Duhok, Iraq, oligoasthenospermia was seen in 20.7% cases.[16] In a study in Kuwait, idiopathic oligospermia was seen in 30% of cases and 50% of the cases were related to testicular causes. In the present study, idiopathic oligoasthenospermia is seen in 34.6%; this result is comparable with that of the Kuwait study.[8] Testicular oligoasthenospermia is seen in 30.7% cases in the present study, whereas in the Kuwait study, it was seen in 50% cases.

Table 6 shows the distribution of the seminal abnormalities in different age groups. In this study, 51.8% of the

Table 5: Distribution of the seminal abnormalities in various groups

	AZO (n = 27)	%	*OLI (n = 8)*	%	*OA (n = 26)*	%	*OAT (n = 32)*	%
Pretesticular	1	3.7	1	12.5	0	–	1	3.1
Testicular	12	44.4	2	25	8	30.7	5	15.6
Posttesticular	7	25.9	1	12.5	9	34.6	7	21.8
Idiopathic	7	25.9	4	50	9	34.6	19	59.3

The percentages are the column percentages;
AZO: Azoospermia; OAT: Oligoasthenoteratospermia;
OA: Oigoasthenospermia; OLI: Oligospermia

Table 6: Distribution of the seminal abnormalities in different age groups

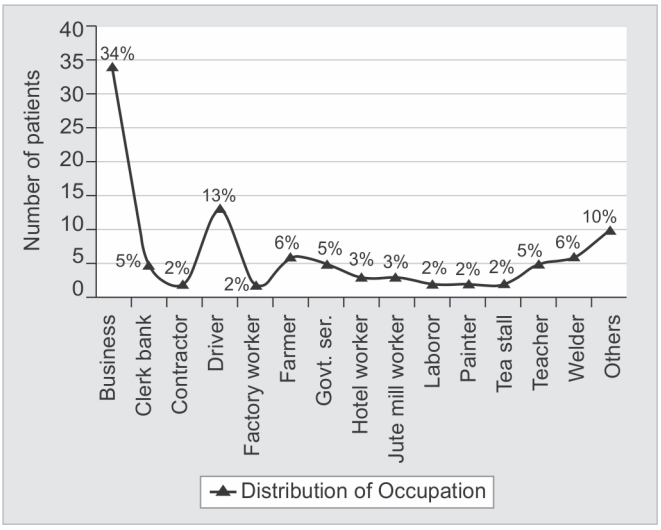
Sl. no.	*Age in years*	*AZO (n = 27)*	%	*OAT (n = 32)*	%	*OA (n = 26)*	%	*OLI (n = 8)*	%
1	<25	1	3.7	–	–	1	3.8	–	–
2	25–35	14	51.8	23	71.8	17	65.3	4	50
3	>35–45	11	40.7	7	21.8	7	26.9	4	50
4	>45–55	1	3.7	2	6.2	1	3.8	–	–

AZO: Azoospermia; OAT: Oligoasthenoteratospermia;
OA: Oligoasthenospermia; OLI: Oligospermia

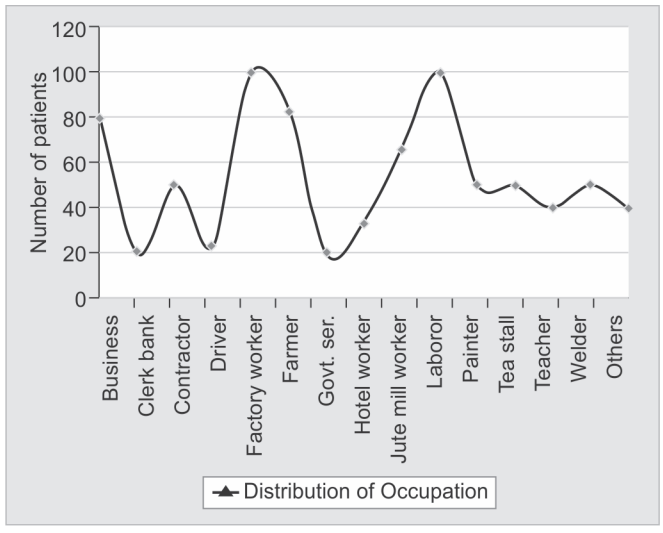
Table 7: Distribution of the seminal abnormalities with duration of infertility

Sl. no.	Duration of infertility (years)	AZO (n = 27)	%	OAT (n = 32)	%	OA (n = 26)	%	OLI (n = 8)	%
1	<5	9	33.3	13	40.6	11	42.3	3	37.5
2	>5–10	14	51.8	14	43.7	11	42.3	3	37.5
3	>10	4	14.8	5	15.6	4	15.3	2	25

AZO: Azoospermia; OAT: Oligoasthenoteratospermia; OA: Oligoasthenospermia; OLI: Oligospermia



Graph 3: The distribution of patients according to occupations



Graph 4: The distribution of smokers among the various occupational groups

azoospermics are in the 25 to 35 years age group, 40.7% in >35 to 45 years, 3.7% in <25 years, and 3.7% in >45 years. But 50% of the oligospermics are in the 25 to 35 year groups and 50% in >35 to 45 year group.

Majority (65.3%) of the OA cases are in the 25 to 35 years age group, 26.9% in >35 to 45 years, and equal numbers in <25 years and >45 years (3.8%). Most of the OAT (71.8%) are in the 25 to 35 years group, 21.8% in >35 to 45 years, and 6.2% in >45 to 55 years.

Table 7 shows the distribution of the seminal abnormalities with the duration of infertility. In this study, 51.8% of the azoospermics had infertility for 5 to 10 years, 33.3% for less than 5 years, and 14.8% for the more than 10 years. A total of 37.5% of oligospermics had infertility for less than 5 years, 37.5% for more than 5 to 10 years, and 25% for more than 10 years. In this study, 42.3% of the OA cases had infertility for less than 5 years, 42.3% for more than 5 but less than 10 years, and 15.3 % for more than 10 years. In this study, 40.6% of OAT had infertility for less than 5 years. A total of 43.75% of the OAT cases had infertility for >5 years but <10 years and 15.6% for more than 10 years. In one study of 66 infertile patients, where the mean age was 35.6 years, azoospermia was seen in 52, asthenozoospermia in 6, oligoasthenospermia in 6, and oligoastheniateratozoospermia in 8.[17]

In the Table 8 study, 29.4% businessmen had OAT, 26.4% had OA, and 14.7% had azoospermia. Among

Table 8: Distribution of seminogram in various occupational groups

	n	OLI	%	OAT	%	OA	%	AZO	%	n	%
Business	34	5	14.7	10	29.4	9	26.4	5	14.7	5	16
Clerk	5	–	–	1	20	–	–	4	80	–	–
Contractor	2	–	–	–	–	–	–	2	100	–	–
Driver	13	1	7.6	6	46.15	3	23.07	3	23.0	–	–
Factory worker	2	–	–	–	–	1	50	1	50	–	–
Farmer	6	–	–	3	50	3	50	–	–	–	–
Govt service	5	–	–	2	40	1	20	1	20	1	20
Hotel worker	3	–	–	1	33.3	1	33.3	1	33.3	–	–
Jute mill worker	1	–	–	–	–	–	–	2	–	–	–
Laborer	2	–	–	1	50	–	–	–	–	1	50
Painter	2	–	–	–	–	–	2	100	–	–	–
Tea stall worker	2	–	–	1	50	–	1	50	–	–	–
Teacher	5	–	–	–	–	3	60	2	40	–	–
Welder	6	0	–	1	16.6	3	50	2	33.3	–	–

The table shows the seminogram distribution in various groups; AZO: Azoospermia; OAT: Oligoasthenoteratospermia; OA: Oligoasthenospermia; OLI: Oligospermia

contractors, 100% had azoospermia; 80% of the clerks, 23% of drivers, and 50% of factory workers had azoospermia; 50% of farmers had OAT and 16.6% of welders had OAT; among teachers, 60% had OA and 40% had azoospermia.

The study of workplace hazards and their effects on the male reproductive function is arguably not new Graphs 3 and 4. The first reports were published in the late 1970s. The very first study, published in 1975, suggested that reproductive function might be impaired in workers exposed to lead. Two years later, another study observed that male workers exposed to the pesticide dibromochloropropane had a high incidence of infertility associated with markedly reduced sperm count.[18] Interest in environmental toxins and male reproduction has also been piqued by recent data demonstrating significant declines in average sperm densities over the past century in men from Western industrialized nations. This is not seen in men from non-Western countries.

Spermatogenesis has been shown to be adversely affected by exposure to lead, mercury, arsenic, hydrocarbons, cadmium, amebic idée soil fumigants, as well as 2-bromopropane, a substitute for chlorofluorocarbons.[19] Exposure to a variety of agricultural pesticides has also been linked to impaired semen quality.[20] Men with exposure to organic solvents have been reported to have elevated serum follicle-stimulating hormone levels as compared with unexposed workers[21] as well as impaired semen parameters.[22] Patients thought to have high exposure to heavy metals, such as lead or mercury, should have serum levels measured if possible.

Most controversial is the theory that sperm counts in men are declining over time, and the reason for this decline is prenatal exposure to environmental toxicants (endocrine disruptors) that have estrogenic effects on the embryo. It is suggested that prenatal exposure to endocrine disruptors cause a testicular dysgenesis syndrome leading to increased rates of cryptorchidism, hypospadias, testicular cancer, as well as infertility.[23] Recent research has demonstrated that chemicals may act as antiandrogens, inhibitors of steroidogenic pathways, as well as estrogens or antiestrogens (Fisher).

The following occupations are at a risk of male infertility: Agriculture and herbicides, construction, machinists, military (Vietnam), plastic production (styrene and acetone), printing industry, service station, mechanics, smiths, taxi drivers, tobacco processing, and welding.

The current scientific literatures say that the following affect seminal parameters aromatic hydrocarbons are involved in low sperm parameters. carbonyl (sevin), ethylene dibromide, ethylene glycol ether, ethylene oxide, impregnates of wood, lead, manganese, metals, organic solvents, paint, pesticides, petrochemicals, radiation, rubber, and chemicals solvents.

Table 9: Distributions of seminogram

Sl. no.	*Type of seminal abnormality*	*n = 100*	*%*
1	Oligospermia	8	8
2	Oligoasthenoteratospermia	32	32
3	Oligoasthenospermia	26	26
4	Azoospermia	27	27
5	Normal	7	7

The following toxins at workplace are considered lethal to male reproductive health: Benzene, bromine, vapor cadmium, carbon disulfide chromium, dibenzofurans, diesel exhaust heat, high-voltage hydrocarbons, kepone, methylene chloride, paint shop solvents, perchloroethylene (dry cleaning), phthalate esters, radar, smoking spray, paint toluene, vibrations, and xylem.[24]

Table 9 shows the distribution of the seminogram. Oligoasthenoteratospermia is the most common abnormality in 32% of the cases, followed by azoospermia in 27%; OA is seen in 26% of patients and oligospermia in 8%. Only 7% of patients had normal sperms.

In one study, oligospermia was seen in 10.4% cases, asthenospermia in 7.3%, teratospermia in 2.1%, obstructive azoospermia in 8.3%, and idiopathic azoospermia in 0.5%. In our study, oligospermia is seen in 8% cases; in a cohort study at Duhok, Iraq, azoospermia was seen in 13% of cases and oligoasthenospermia in 20.7%.[25] In our study, azoospermia was seen in 27% and oligoasthenospermia in 26%.

In another study, asthenospermia was seen in 6% cases, teratospermia in 4%, and oligospermia in 4%. In our study, oligospermia was seen in 8% cases.

In the Kuwait study, 38% had azoospermia and 62% had oligospermia. In our study, azoospermia was seen in 27%.

In the Mongolian study, azoospermia was seen in 20.5%, astheniaspermia in 7.4%, abnormal seminal plasma in 3.75%, teratospermia in 1.2%, and oligospermia in 11.6%.

In Nigeria, 3.1% were azoospermic, 15.4% oligospermic, and 4.3% teratozoospermic. Some of the abnormal seminal samples demonstrated combined defects with oligoasthenospermia occurring in 10% of the entire study population;[25] in another study of a total of 209 patients, 145 (29%) were oligospermic, 59 (11.8%) severe oligospermic, and 87 (17.4%) azoospermic.

In the Japan study, the incidence of azoospermia was 44.7%.

CONCLUSION

Male infertility is multifactorial. Age, occupation, and habits have a significant impact on the seminal parameters. Modifiable behaviors like cessation of smoking and alcohol are cost-effective in normalizing

the semen parameters and thereby restoring fertility. The mean age group is 34.89 years (minimum age group is 22 years and maximum age group is 52 years), and the mean duration of infertility is 6.95 years (minimum is 2 years, maximum duration is 16 years).

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