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Research Article

Substance Abuse and Sperm Count Amongst Patients Attending Semen Analysis Laboratory at Tertiary Care Centre

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Abstract: The aim of present study was to find relation between smoking, alcohol consumption & smokeless tobacco consumption and sperm count. The study was carried out on 250 consenting males attending the semen analysis laboratory of the tertiary care centre from January 2014 to August 2014. After recording the demographic data, history of tobacco use in the form of smoking and smokeless tobacco and alcohol consumption was taken. Semen samples were categorised into normospermia, oligospermia, and azoospermia. Proportion of azoospermic and oligospermic males was 10.8% (27) and 18.4% (46) respectively. Low sperm count was significantly associated with smoking (odds ratio 1.93, 95% Confidence Interval 1.08-3.44) (p value 0.02). Low sperm count was not significantly associated with Alcohol consumption & smokeless tobacco consumption. Smoking is a significantly associated with low sperm count.
Keywords: Smoking, Alcohol, Smokeless tobacco, Sperm count.

INTRODUCTION

In addition to the high population growth in many developing countries, it has been noticed that infertility is on the rise in the reproductive age. Approximately about 10-15% of couples suffer from infertility all over the world. Female factor is responsible in 35% and male factor in 45% of cases and remaining couples either have combination of factors or unexplained infertility [1]. Male infertility has been suggested to be an important but neglected reproductive health issue [2]. The pathological causes for decreased sperm count is due to the abnormality in the control mechanism of sperm production at pre-testicular, testicular or post testicular level [3].

Studies from several populations around the world indicated that smoking, types of occupation, alcohol consumption and coffee intake and nutritional status are risk factors of male infertility [4-6]. Cigarette smoking has been widely recognized as a health hazard. Despite worldwide antismoking campaigns, some people consume cigarettes on a regular basis. The highest prevalence of smoking is observed in young adult males during their reproductive period [7]. Similarly alcohol drinking & tobacco chewing are also recognised as health hazard.

Semen analysis remains the single most useful and fundamental investigation for searching the cause of male infertility. It assesses the formation and maturity of sperm and the interaction of the sperm with the seminal fluid, thus providinginsight on sperm production (count) and sperm quality (motility, morphology) [8, 9]. Screening by semen analysis provides us with a baseline before going for extensive investigation.

The aim of the study was to determine the likely risk factors, such as smoking, alcohol consumption & smokeless tobacco consumption on male infertility and to study the effects of the semen parameters, including volume, motility, viability and normal morphology, in male infertility

METHODOLOGY

The present study was carried out at the Department of Physiology, Government Medical College & Hospital, Nagpur from January 2014 to August 2014. A total 250 consenting male attending the semen analysis laboratory of the hospital between the ages of 20 and 40 years were recruited. Detailed history was taken from the participants regarding age, duration of marriage, occupation, smoking, alcohol consumption, smokeless tobacco consumption, sexual history, infertility (primary or secondary infertility),

first or second marriage, drugs, surgical and medical history for any illness. Males who did not give consent and those who were unable to pass specimen by masturbation were excluded.

Male participant were instructed to keep abstinence from coitus for 3-4 days before attending the laboratory and procedure to collect the samples was explained in detail. Samples were collected aseptically by masturbation into sterile wide-mouthed bottles within hospital premises. Semen analysis was performed according to the methods and standards outlined by the World Health Organisation (WHO) [10]. Semen analysis was performed within 60 minutes of sample collection for volume, concentration, motility, morphology, viability and the presence of pus cells. Semen volume was measured with a graduated disposable pipette. After liquefaction, the semen specimen was thoroughly mixed and a drop was spread on a glass slide by placing a cover slip on it. Sperm motility was assessed by microscope appraisal of spermatozoa from different fields.

Semen samples were categorised on the basis of sperm count per ml of semen in accordance with WHO normal and pathological ranges i.e. normospermia, oligospermia, and azoospermia. Parameters outlined included: Volume: 2.0ml or more; Sperm concentration: $>15x10^6$ spermatozoa/ml; Total sperm count: $39x10^6$ per ejaculate or more; Motility: 50% or more with forward progression [9].

The samples categorised were compared for ejaculated volume, pus cells, motility and morphology. The following operational definitions were used: Normospermia: Sperm count 20 million/ml & above; Oligospermia: Sperm count below 20 million/ml; Azoospermia: Absence of spermatozoa in the ejaculation [9]. Mean ± Standard deviation (SD) were calculated for sperm count, volume, pus cells, motility; Mean values were compared for statistical significance using t-value with level of significance <0.05 (p value).

Based on history of smoking, study subjects were classified as: non-smoker (individuals who never smoked tobacco in any form before); smoker (anyone who was currently smoking tobacco in any form).

Based on history of alcohol consumption, study subjects were classified as: Lifetime abstainer - has had fewer than 12 drinks in entire lifetime. Drinker - has had at least 12 drinks in lifetime and at least one drink in the past year.

Smokeless tobacco addiction is defined as tobacco that is not smoked but used in another form such as chewing tobacco or snuff. Based on history of smokeless tobacco consumption, study subjects were classified as: non- addicted (individuals who never used smokeless tobacco before); addicted (anyone who was currently using smokeless tobacco).

History of smoking, alcohol drinking & smokeless tobacco use among normospermic, oligospermic & azoospermic subjects was compared for statistical significance using chi square test, with level of significance <0.05 (p value).

RESULTS

Of the total, 270 males who were willing to give semen sample for analysis, 20 were unable to pass specimen. Among the 250 males, the mean age was 28±5.34 years; 210 (84%) males had primary infertility, and 40 (16%) suffered from secondary infertility. Mean duration of infertility was 4.56±3.85 years. Using WHO standard for semen normality, 250 samples were analyzed. Out of that normal sperm count (normospermia) was observed in 177 males (70.8 %), Oligospermia was observed in 46 males (18.4%) & azoospermia was observed in 27 males (10.8%) (Table 1).

After excluding 27 samples with azoospermia, semen parameters were compared in oligospermic and normospermic samples for count, volume, pus cell, motility and morphology (Table 2). The mean sperm count in normospermic males was 145.72 $\pm$ 72.35 millions and mean sperm count in oligospermic males was 5.67 $\pm$ 4.73 millions. The oligospermic samples had significantly lower percentage of motile sperms 37.84 ± 22.45 compared to normospermia in which motile sperms were 59.23 ± 18.70%, (p <0.0001). Comparison of volume showed mean volume of 2.95 $\pm$ 1.34ml in normospermia versus 2.04 $\pm$ 1.98ml in oligospermia (p <0.0047), and pus cells 8.10 $\pm$ 7.7 in normospermia versus 12.07 $\pm$ 10.23 in oligospermia, (p = 0.0167).

We compared the history of smoking, alcohol consumption & smokeless tobacco consumption in normospermic, oligospermic & azoospermic subjects (Table 3). Oligospermia and azoospermia was significantly higher among those who had history of smoking (p=0.02). Those who had history of smoking were at 1.93 (95% CI 1.08-3.44) times higher risk of oligospermia and azoospermia. The proportion of oligospermia and azoospermia was higher among those who had history of alcohol and tobacco consumption as compared to those who had no history of alcohol and tobacco consumption; but this difference was not statistically significant.

Table 1: Distribution of study subject on the basis of sperm count

Category	No=250	Percentage %
Normospermia	177	70.8
Oligospermia	46	18.4
Azoospermia	27	10.8

Table 2: Comparison of semen parameters between normospermia and oligospermia

Parameters	Normospermia Mean ± SD	Oligospermia Mean ± SD	p value
Count(millions)	145.72 ± 72.35	5.67 ± 4.73	<0.0001
Volume (ml)	2.95 ± 1.34	2.04 ± 1.98	0.0047
Pus Cells	8.10 ± 7.7	12.07 ± 10.23	0.0167
Motile Sperm (%)	59.23 ± 18.70	37.84 ± 22.45	<0.0001

Table 3: Distribution of study subjects by type of addiction

Type of addiction	Normospermia (n=177)	Oligospermia + Azoospermia (n = 46 + 27 = 73)	OR (95% CI)	p value
Smoking				
Yes	45	19+10 = 29	1.93 (1.08-3.44)	0.02
No	132	27+17 = 44		
Alcohol Consumption				
Yes	68	21+8 = 29	1.05 (0.6-1.84)	0.84
No	109	25+19 = 44		
Smokeless Tobacco				
Yes	34	13+9 = 22	1.81 (0.9-3.38)	0.06
No	143	33+18 = 51		

DISCUSSION

There has been an increase in the cases of male infertility from all over the world and similar trend is observed in India. Screening of males by semen analysis provides some insight about the underlying pathological problems occurring in the male genital tract. The objective of this study was to determine factors associated with male infertility in tertiary care centre, in central India.

In our study proportion of normospermic males was found to be 70.8% that of oligospermic males & azoospermic was 18.4% & 10.8% respectively. The results are comparable with a study done by Fauzia Butt *et al*. [9], in which they found normospermia in 73.99% males, oligospermia in 11.11% & azoospermia in 14.89%.

In the present study mean sperm count in normospermic males was 145.72 ± 72.35 millions and that in oligospermic males was 5.67 ± 4.73 millions. The oligospermic samples had significantly lower percentage of motile sperms 37.84 ± 22.45 compared to normospermia in which motile sperms were 59.23 ± 18.70%, (p <0.0001). Comparison of volume showed mean volume of 2.95 ± 1.34ml in normospermia versus 2.04 ± 1.98ml in oligospermia (p =0.0047), and pus cells 8.10 ± 7.7 in normospermia versus 12.07 ± 10.23 in oligospermia, (p = 0.0167). Similar findings were noted by Mortimer D *et al*. [11] in their study.

In our study smoking was significantly associated with decreased sperm count (oligospermia & azoospermia) (P=0.02). Findings are comparable with study done by Sin-Eng Chia *et al*. (OR 2.82; 95% CI 1.93413) [12] and G. Collodel *et al*. (p <0.05) [12]. Although present study being a cross sectional study cannot prove the causal relationship, a causal relationship between cigarette smoking and impaired reproductive function is highly suspected in the literature [13]. Results of many studies show a substantial negative effect of cigarette smoking on sperm production, motility, and morphology [14, 15]. Several studies have reported that the mutagenic components of cigarette smoke adversely affect rapidly dividing cells, including germ cells in the testis [16].

In our study alcohol consumption was not found to be significantly associated with decreased sperm count (oligospermia & azoospermia) (p=0.84). Similar findings were present in study done by Sin-Eng Chia *et al*. [12]. However in the study done by K. R. Muthusami *et al*. [17] it was found to be significantly associated with it. The reason may be long duration of their study.

In our study smokeless tobacco consumption was not found to be significantly associated with decreased sperm count (oligospermia & azoospermia) (p=0.06). This is contrary to the findings of Tamer M. *et al*. [18] and Sangita Phatale *et al*. [19]. It may be due to larger sample size in their study.

CONCLUSION

In this study it was found that, 18.4% study subjects were oligospermic and 10.8% were azoospermic. Decreased sperm count was significantly higher among subjects with history of smoking.

REFERENCES

1. Carlsen E, Giwercman A, Keiding N, Skakkebaek NE; Evidence for decreasing quality of semen during past 50 years. BMJ, 1992; 305(6854): 609-613.
2. Dhont N; Clinical, epidemiological and socio-cultural aspects of infertility in resource-poor settings. Evidence from Rwanda. Facts Views Vis Obgyn., 2011; 3(2): 77–88.
3. Acacio BD, Gottfried T, Israel R, Sokol RZ; Evaluation of a large cohort of men presenting for a screening semen analysis. Fertil Steril., 2000; 73(3): 595-597.
4. Chia SE, Lim ST, Tay SK, Lim ST; Factors associated withmale infertility: a case control study of 218 infertile and 240 fertile men. BJOG, 2000; 107(1): 55–61.
5. Bayasgalan G, Naranbat D, Radnaabazar J, Lhagvasuren T, Rowe PJ; Male infertility: risk factors in Mongolian men. Asian J Androl., 2004; 6(4): 305–311.
6. Petrelli G, Mantrovani A; Environmental risk factors and male infertility and reproduction. Contraception, 2002; 65(4): 297–300.
7. Langgassner J; Rauchgewohnheiten der osterreichischen Bevolkerung. Satistische Nachr., 1999; 5: 319–326.
8. Harry F; Declining worldwide sperm counts: Disproving a myth. Urol Clin N Am., 2008; 35(2): 137-146.
9. Butt F, Akram N. Semen analysis parameters: Experiences and insight into male infertility at a tertiary care hospital in Punjab. J Pak Med Assoc., 2013; 63(5): 558-562.
10. WHO; Laboratory manual for the examination and processing of human semen. 5th edition. Available from http://www.who.int/reproductivehealth/publications/infertility/9789241547789/en/
11. Mortimer D, Templeton AA, Lenton EA, Coleman RA; Semen analysis parameters and their interrelationships in suspected infertile men. Arch Androl., 1982; 8(3): 165-171.
12. Chia SE, Lim ST, Tay SK, Lim ST; Factors associated with male infertility: a case-control study of 218 infertile and 240 fertile men. BJOG, 2000; 107(1): 55-61.
13. Collodel G, Capitani S, Pammolli A, Giannerini V, Geminiani M, Moretti E; Semen quality of male idiopathic infertile smokers and nonsmokers: An ultrastructural study. J Androl., 2010; 31(2): 108-113.
14. Weber RF, Pierik FH, Dohle GR, Burdorf A; Environmental influences on male reproduction. BJU Int., 2002; 89(2): 143–148.
15. Kunzle R, Mueller M, Ha¨nggi W, Birkha¨user M, Drescher H, Bersinger N; Semen quality of male smokers and nonsmokers in infertile couples. Fertil Steril., 2003; 79(2): 287-291.
16. Sorahan T, Prior P, Lancashire RJ, Faux SP, Hulte´n MA, Peck IM *et al.*; Childhood cancer and parental use of tobacco: deaths from 1971 to 1976. Br J Cancer, 1997; 76(11): 1525–1531.
17. Muthusami KR, Chinnaswamy P; Effect of chronic alcoholism on male fertility hormones and semen quality. Fertil Steril., 2005; 84(4): 919-924.
18. Said TM, Ranga G, Agarwal A; Relationship between semen quality and tobacco chewing in men undergoing infertility evaluation. Fertil Steril., 2005; 84(3): 649-653.
19. Phatale SR, Boramma S; Effect of tobacco chewing and smoking on male infertility. International Journal of Recent Trends in Science and Technology, 2014; 9(3): 386-388.