Int J Pharm Bio Sci 2013 Oct; 4(4): (B) 513 - 518

Research Article **Pharmacology**

International Journal of Pharma and Bio Sciences **ISSN 0975-6299**

IMPACT OF CIGARETTE SMOKING AND ALCOHOL INTAKE ON MALE INFERTILITY

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ABSTRACT

Chronic alcohol consumption and cigarette smoking adversely affect male reproductive health. This work highlights the influence of both on the sperm quality of infertile male partners. This prospective study was conducted on semen samples of alcoholics-(Group1), smokers-(Group2), habit of both-(Group-3), controls-(Group4). Samples were analysed following WHO guidelines including the percentage of teratoszoospermia, asthenozoospermia , oligospermia and their combination. F-test was done between alcoholics, smokers, and both smokers and alcoholics with controls using SPSS. Teratozoospermia and asthenozoospermia dominated in Group1 whereas asthenozoospermia and oligospermia dominated in Group2. Oligospermia dominated in group 3 and as the age advances all the three variables dominated in group2 and 3. Alcohol intake affects sperm morphology and motility, whereas smoking affects sperm concentration and motility. Progressive deterioration in semen quality is related to increasing quantity of cigarette smoking than alcohol intake.

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INTRODUCTION

Infertility, a basic disease of the reproductive system affects 10-15% of all couples [1]. Even though male infertility plays a key role in conception, in 40% of infertile couples no apparent reason for infertility could be found. This has drawn attention to the impact of life style and environmental factors especially diet, obesity, smoking, alcohol intake, recreational drug use and exposure to environmental toxins on reproductive health[2]. Cigarette smoking and alcohol abuse are widely recognized as health hazard worldwide every year. Smoking and alcohol consumption are frequently associated with impaired male reproductive function. Many workers have examined the effects of tobacco smoking and alcohol consumption on human seminal quality. Smoking adversely affects male reproductive health and chronic alcohol consumption has been associated with abnormal sperm morphology. Cigarette smoking is related with decreased sperm concentration, decreased sperm motility and a reduced percentage of morphological normal sperm and impaired semen parameters [3,4,5]. The objective of the present study is to analyse the distribution of sperm quality due to alcohol consumption and cigarette smoking and their frequency on randomly selected infertile men attending the andrology laboratory for treatment.

MATERIALS AND METHOD

Male partners of infertile couples seeking treatment for primary infertility were included in this study. This prospective study was conducted at andrology laboratory of Prashanth fertility centre and Sree Balaji Medical College after getting institutional ethical clearance of Sree Balaji medical college and hospital and informed consents from the subjects. Married more than one year and not using any contraceptive measures were selected for this study. In this study the effect of alcohol and smoking and its synergistic effect on semen quality in four different groups were analyzed. The subjects consuming alcohol for a period of more than three years were considered as alcoholics-(Group1).Subjects without alcohol consumption but with cigarette smoking more than a period of three years were considered as smokers –(Group2). Both alcoholics and smokers were included as (Group3). Strict non alcohol consumers and non smokers were selected as controls- (Group4).

EXCLUSION CRITERIA

1) History of prolonged medication and intake of herbal /tonics and indigenous medicine.
2) Varicocele, hydrocele, undescented testis, vasectomy reversal surgery and history of chronic illness.
3) Males above 45yrs of age
4) Those with negative semen fructose test.

SAMPLE COLLECTION

Samples were collected in sterile containers by masturbation in the laboratory following strict abstinence for four to six days. All samples were kept at $37^{o}\pm 2^{0}$ c temperature and processed immediately after complete liquefaction. All semen samples were analyzed for 10 primary semen parameters.1)Liquefaction time 2)Volume 3)Viscosity 4) Amorphous particulate matter 5) Agglutination 6)Motility 7)Viability 8)sperm density 9) Morphology (normal forms) and 10) Head less spermatozoa. The results of semen analysis were classified according to the nomenclature of semen variables (World health organization 1992).The samples is considered Normozoospermia when sperm concentration, motility and morphology were within reference values. The reference value of sperm concentration was greater than $20x10^{6}$ The motility was assessed using sperm progression rating. A- Rapid forward progressive motility, B- Slow or sluggish progressive motility, C- Non progressive motility and D- Immotility. Motility was defined as normal when greater than 50% of sperm were classified as grade A+B or greater than 25% were grade A, Teratozoospermia was determined when sperm morphology was less than the reference value, like wise asthenozoospermia was diagnosed when motility and oligzooospermia when sperm concentration

were below the reference value. When all the three variables like concentration, motility, morphology were disturbed an oligoasthenoteratospermia was diagnosed, Azoospermia was diagnosed when even in the sediment after centrifugation at greater than 3000g for 15 min no sperm were detected.Combinations like oligoteratozoospermia,oligoasthenozoospermia, and asthenoteratozoospermia were used when two variables were disturbed.

RESULTS

In this study 524 men were evaluated for infertility, of those 159 were alcoholics, 154 smokers, 130 were both alcoholics and smokers and 81 were non smokers and non alcoholics. The contribution of three main semen variables viz Teratozoospermia, asthenozoospermia and oligospermia or their combination which we observed during semen analysis in four groups were shown in tables. Teratozoospermia and asthenozoospermia are the dominated semen variables and also in various combination like (T+A) (A+T+O) in Group1,and progressive increase in Teratospermia was noticed as the duration of the alcoholism and age of the individuals increases, thus alcoholics showed a defect in motility and morphology as shown in table5. Asthenzoospermia and oligospermia dominated the semen variables individually or in various combinations in smokers (Group2). In this group (A+O+T) were noticed in large numbers as the age and of the smoking increases. Thus smokers showed a defect in sperm count and motility as shown in table 5. In alcoholics and smokers (Group3) oligospermia was noticed predominantly and defect in sperm count was noticed in higher number as shown in table 5. In controls (Group4) teratozoopermia is seen in most cases as shown in table 5.

TABLE 1
SEMEN VARIABLES AMONG ALCOHOL CONSUMERS GROUP 1

	AGE		
	25-30 N(%)	31-35 N(%)	36-40 N(%)
T	8(18.6)	8(16.3)	13(22.3)
T+O	5(11.6)	7(14.2)	7(10.4)
T+A	6(13.9)	7(14.2)	10(14.9)
A	6(13.9)	7(14.2)	12(17.9)
A+O	7(16.2)	8(16.3)	8(11.9)
O	7(16.2)	7(14.2)	8(11.9)
OAT	4(9.3)	5(10.2)	7(10.4)

TABLE 2
SEMEN VARIABLES AMONG SMOKERS GROUP 2

	AGE		
	25-30 N(%)	31-35 N(%)	36-40 N(%)
T	7(14.8)	5(10.4)	5(8.4)
T+O	5(10.6)	6(12.5)	8(13.5)
T+A	6(12.7)	6(12.5)	7(11.8)
A	714.8)	8(16.6)	10(16.9)
A+O	7(14.8)	7(14.5)	8(13.5)
O	8(17)	7(14.5)	12(20.3)
OAT	7(14.8)	9(18.7)	9(15.2)

TABLE 3
SEMEN VARIABLES AMONG SMOKERS AND ALCOHOL GROUP 3

	AGE		
	25-30 N(%)	31-35 N(%)	36-40 N(%)
T	5(11.9)	8(14.5)	5(15.1)
T+O	5(11.9)	6(10.9)	4(12.1)
T+A	4(9.5)	5(9.0)	3(9.0)
A	7(16.6)	9(16.3)	6(18.1)
A+O	6(14.2)	8(14.5)	4(12.1)
O	8(19)	10(18.1)	6(18.1)
OAT	7(16.6)	9(16.3)	6(18.1)

TABLE 4
SEMEN VARIABLES AMONG CONTROLS GROUP 4

	AGE		
	25-30 N(%)	31-35 N(%)	36-40 N(%)
T	5(22.7)	6(230	9(28.1)
T+O	3(13.6)	3(11.5)	4(12.5)
T+A	3(13.6)	3(11.5)	3(9.3)
A	4(18.1)	5(19.2)	6(18.7)
A+O	4(18.1)	4(15.3)	5(15.6)
O	3(13.6)	5(19.2)	5(15.6)
OAT	0	0	0

TABLE 5
COMPARISON OF SEMEN VARIABLES OF ALCOHOL, SMOKERS, BOTH CONSUMERS WITH CONTROLS

ALCOHOLICS

	25-30	31-35	36-40	TOTAL	CONTROL	P-VALUE
Teratozoospermia (T)(A+T)(O+T)(A+O+T)	23	27	39	89	39	<0.0001
Asthenospermia (A)(A+T)(A+O)(A+O+T)	23	27	37	87	37	0.0009
Oligospermia (O)(O+A)(O+T)(A+O+T)	23	27	30	80	39	0.0232

SMOKERS

	25-30	31-35	36-40	TOTAL	CONTROL	P-VALUE
Teratozoospermia (T)(A+T)(O+T)(A+O+T)	25	26	29	80	39	0.0321
Asthenospermia (A)(A+T)(A+O)(A+O+T)	27	30	34	91	37	0.0008
Oligospermia (O)(O+A)(O+T)(A+O+T)	27	29	37	93	36	0.0007

BOTH Alcoholics and smokers

	25-30	31-35	36-40	TOTAL	CONTROL	P-VALUE
Teratozoospermia (T)(A+T)(O+T)(A+O+T)	21	28	18	67	39	0.0021
Asthenospermia (A)(A+T)(A+O)(A+O+T)	24	31	19	74	37	0.0035
Oligospermia (O)(O+A)(O+T)(A+O+T)	26	33	20	79	36	0.0008

T = TERATOZOOSPERMIA
A = ASTHENOZOOSPERMIA
O = OLIGOSPERMIA
P VALUE < 0.0001 SIGNIFICANT

DISCUSSION

Hypothalamo –pituitary gonadal axis is interfered by alcohol intake and has been shown to have a deleterious effect at all levels on male reproductive system. It has been reported to cause impaired testosterone production which results in impotence, infertility and reduced male secondary sexual characteristics[6,7,8]. It also results in impaired production and secretion of luteinizing hormone and follicle stimulating hormone leading to deterioration of sertoli cells[9]. Of the semen parameters analyzed among alcoholics,

teratospermia and asthenospermia were found dominant among the semen variables. The sperm abnormality with motility defect was observed more as the age advances in alcoholics. Some of the protein required for sperm cell production is probably damaged by alcohol[10] which induce reduction in level of testosterone, LH, and FSH which hampers their normal morphological development and maturation of spermatozoa production and significantly leads to teratospermia. It also slows down the sperm motility especially as the age advance and produce chronic ailment on production of testicular germ cells[9]. Asthenozoospermia and oligoszoopermia dominated among the semen variables individually or in various combinations in smokers. In this group (A+O+T) were noticed in large numbers with increasing age of a person and duration of smoking. The toxins in cigarette reach male reproductive organ, interact with seminal fluid components and the accessory glands leading to increased viscosity, reduced seminal volume and delay the liquefaction time, and hence reducing forward linear progression of spermatozoa, manifesting as asthenozoospermia[11,12,13,14,15]. Exposure of spermatozoa to the toxins in cigarette smoke probably tilts the delicate balance of reactive oxygen species (ROS) that are produced by spermatozoa for their special functions like decapitation. Increased quantities of ROS have been shown to be detrimental to the DNA of spermatozoa, thus producing a negative effect on the viability and morphology of spermatozoa[16]. The presence of all the three variables astheno, oligo, as well as teratozoopermia had a significant contribution in the development of morphological abnormalities and reduced sperm counts besides motility defects[13, 14,17]. The mean reduction in sperm concentration by 13%, a mean reduction of sperm motility by 10% and reduction of morphological normal sperm by 3% was reported in smokers[18]. Chronic smoking leads to degeneration of leydig cells which in turn reduces testosterone production, which has proved in rats when exposed to cigarette smoke[19]. In a data published by IIT Madras, the oxides of nitrogen and carbon monoxide concentrations in the ambient air have been steadily increasing in Chennai from 1993 to 2006.Transport is the source of CO, NO_2 and also lead in Chennai because of increasing number of vehicles in recent years was the finding carried out on behalf of a leading daily (The Telegraph). The Tamil Nadu pollution board also substantiates the fact that suspended particulate matter (SPM), respirable particulate matter (RPM) and oxides of nitrogen levels were above the normal range in certain regions of Chennai. A pilot study also revealed that fruits and vegetables grown on land irrigated with water that carries untreated sewage in Chennai were contaminated with lead, cadmium and nickel. The anti androgenic effect of the environmental toxicants and xenoestrogens may have a detrimental effect on the functioning of sex glands, affecting the sperm motility parameters. This work is biased, as the above factors cannot be ruled out in assessing the quality of semen.

CONCLUSION

It has been proved by various studies that smoking or alcohol consumption either alone or together affects the sperm parameters in many ways. Our study also is consistent with the above finding. Infertility due to male factor is also equally important while considering the causes for infertility. Since smoking and alcohol habits affecting sperm motility parameters are modifiable risk factors, measures should be taken strictly to warn the individuals against the consumption of both, so as to reduce the occurrence of infertility due to male factors.

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