



MALE INFERTILITY AND ITS CAUSES: AN ANTHROPOLOGICAL PERSPECTIVE

G Sudha and KSN Reddy

Department of Anthropology, Sri Venkateswara University, Tirupathi-517 502, Andhra Pradesh, India.

Corresponding Author: -**KSN Reddy**

E-mail: katarisnr@yahoo.co.in

Article Info

Received 25/05/2014

Revised 18/06/2014

Accepted 20/06/2014

Key words: Male Infertility, Oligospermia, Azoospermia. BMI. Healthy life style.

ABSTRACT

In the present study a total of 523 males were screened to evaluate male infertility and specific causative factors from three infertility centers who are taking medical counseling in Tirupati town, Andhra Pradesh, India. A validated questionnaire has been administered to collect the data pertaining to the causes of infertility, period of infertility and life styles besides anthropometric measurements. Clinical investigations were also carried out to the sample. The period of infertility was divided into five infertile age groups i.e. 1-2 years, 3-4 years, 5-9 years, 10-14 years and >15 years. In 1-2 years of infertile group, 43.29% of Oligospermia; 47.91% of Azoospermia and 66.66% of pre mature ejaculation. In >15 years period of infertile age group, 4.74% of Oligospermia; Azoospermia with 4.16% and pre mature ejaculation was 3.41%. Infertility period increases, BMI also increases. Higher percentage (48.94%) with BMI 25.42 ± 2.1 were present in 1-2 years of infertility period. In 15+ years of infertility period low percentage was observed (4.40%) with high BMI (28.66 ± 3.6). When the infertility cases were rated based on the issues, around 25% males needed medical aid, 70% were normal and only 5% are refractory in nature. 96% of males were found to be habituated to drinking (24%), smoking (33%) and both (39%) habits. In conclusion male adiposity and lifestyle habits were associated with increased infertility in males. Increased adiposity could produce other biological changes in men that reduce their fertility. Weight loss may improve their chances of conception.

INTRODUCTION

Male infertility refers to the inability of a male to achieve pregnancy in a fertile female. Male infertility is considered when identifiable female causes of infertility are excluded and semen quantity and quality fails to fulfill WHO criteria [1]. It is a worldwide problem affecting people of all communities, though the cause and magnitude may vary with geographical location. Infertility is not merely a health problem; it is also a matter of social injustice and inequality [2]. The exact cause of male infertility is still unknown in more than 50% of cases [3]. Approximately one-third of the cases of infertility affecting couples are primarily attributable to the woman, one-third

to the man, one third to an interaction between the two and 20% of those remaining unexplained [4]. Although many people still think of infertility as a "woman's problem", up to half of all cases of infertility involve problems with the man. In fact, in about 20 to 30 per cent of the time, a man's low fertility is the main obstacle to conception. A variety of disorders ranging from hormonal disturbances to physical problems and psychological problems can cause male infertility.

Male infertility is commonly due to deficiencies in the semen and semen quality is used as a surrogate measure of male fecundity [5]. Male infertility is a multifactorial disease process with a number of potential



contributing causes. Male infertility cases are due to deficient sperm production of unknown origin, environmental and nutritional factors. Lifestyle risk factors like cigarette smoking, alcohol consumption, chronic stress, and nutritional deficiencies has been associated with decreased sperm count, alterations in motility, and an overall increase in the number of abnormal sperm [6-8]. Most studies that included alcohol as a point of investigation have failed to show a significant impact on sperm counts, at least among those with moderate alcohol Consumption [9,10]. In contrast, in chronic alcoholics, there is good evidence for impairment of spermatogenesis and reductions in sperm counts and testosterone levels [11,12].

Excess weight is not only linked to increased risk of chronic disease [13], but has also been shown to increase risk of reproductive problems [14]. Excess weight can affect male hormone levels [15-17]. A significantly reduced testosterone to estradiol ratio has been observed among overweight or obese men (BMI>25) when compared with men with lower BMI. Men with higher BMI have also exhibited altered quantity and quality of sperm [18,19]. In the light of above background the present study is intended to cover all aspects of male infertility by considering samples in infertility centers to examine the extent of infertility in Tirupati town of Chittoor district, Andhra Pradesh.

MATERIALS AND METHODS

This is a cross-sectional descriptive study in which 523 infertile men were selected by convenience sampling and were evaluated. The study materials belong to infertile men from all communities of different socio-economic milieu who sought medical assistance in three private infertility Centers available in Tirupati town for treatment. These centers have adequate infrastructural facilities for all kinds of clinical investigations of infertility either of male or female. The purpose and overview of the study was explained at the time of the interview, and interviewees were informed that their participation was entirely voluntary, their anonymity would be assured, they could withdraw from the study at any time and the information that they will be providing would be used solely for the purposes of the study. They were also told that the researcher would assume responsibility for the safekeeping of the data, and that they could request deletion of their data at any point. The exclusion criteria are any physical illness which prevents them from conceiving and suffering from any neurological or psychiatric illness.

A validated questionnaire has been administered to collect the data pertaining to the causes of infertility, period of infertility, life styles besides anthropometric measurements. Clinical investigations were also carried out to the sample. The Anthropometric measurements like height, weight from infertile males were recorded following the procedures of Weiner and Lourie [20] and

WHO [21]. In this paper we followed the BMI classification proposed by the WHO Western Pacific Regional office in collaboration with IOTF [22]. The results thus achieved have been critically analyzed and presented.

RESULTS

A total of 523 infertile males were studied and the sample was arranged according to period of infertility. The results of the present study were presented in four tables. Distribution of the sample according to the nature of disorders and period of infertility was shown in table 1. In a total of 523 infertile males mainly oligospermia (68.45%) dominates over other disorders like azoospermia (9.17%) and pre mature ejaculation (22.37%). Percentage of all disorders was more in the initial period of infertility.

Table 2 shows the relationship between the period of infertility with weight, height and the BMI. It is evident from the table that when the period of infertility increases the mean weight increases whereas the mean height decreases. It is clear from the table that when the infertility period increases, BMI also increases. Higher percentage (48.94%) with BMI 25.42 ± 2.1 are present in 1-2 years of infertility period. In 15+ years of infertility period low percentage was observed (4.40%) with high BMI (28.66 ± 3.6). It shows that when the infertility period and BMI increases, the percentage of infertile individuals decreases. As a result of this it shows that there is a possible association with obesity and infertility.

Classification of infertile males according to the period of infertility and BMI categories are described in table 3. It is observed that more percentage (35.15%) are infertile in 1-2 years of infertility period is present at risk of obesity (23-24.9) category, which is followed by both normal and obese-I categories with the same percentage (21.48%). Further, infertility is greater in 15+ years of infertility period in obese-II (34.78%) individuals followed by obese-I (30.43%) individuals. It is clear from the table that in all infertility periods, higher percent of infertile males are at risk of obese categories and followed by normal and underweight categories.

Table 4 shows the distribution of sample according to type of habits. On the basis of smoking and alcohol habits among the infertile males, it is observed that more percentage (39.19%) of males is infertile when they have both the habits of smoking and alcohol. But when consider the habit of smoking and alcoholism alone, the infertility in smokers is 32.50% and among alcoholics it is 23.90%. It proves that there is a strong association between infertility and smoking and alcoholic habits.

The distribution of male infertility cases by rating (fig: 1) shows that a majority of the cases are normal (70%), while 25% need medical aid. The nature of physiological problems in males is pre mature ejaculation (5%), oligospermia (15%) and azoospermia (5%). Thus the remaining 5% cases are total refractory in nature.



Fig 1. Rating in 100% of male infertility

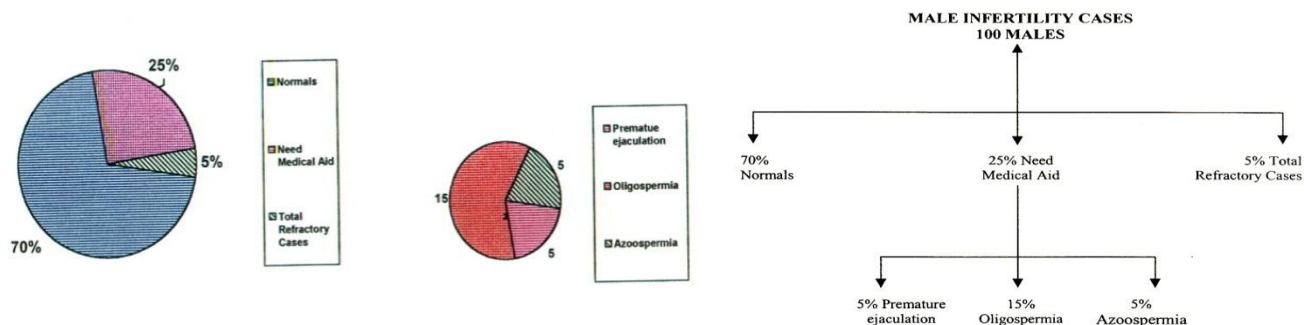


Table 1. Distribution of the sample according to nature of disorders and period of infertility

Period of Infertility (in years)	Nature of Disorders					
	Oligospermia		Azoospermia		Pre mature ejaculation	
	No	%	No	%	No	%
1-2	155	43.29	23	47.91	78	66.66
3-4	103	28.77	14	29.16	20	17.09
5-9	48	13.40	3	6.25	7	5.98
10-14	35	9.77	6	12.5	8	6.83
15+	17	4.74	2	4.16	4	3.41
Total (523)	358	68.45	48	9.17	117	22.37

Table 2. Distribution of the sample by mean weight, height and Body Mass Index (BMI) of infertile males

Infertility Period	Males (523)		Weight±S.D	Height±S.D	BMI±S.D
	No	%			
1-2	256	48.94	74.5±8.7	171.3±7.2	25.42±2.1
3-4	137	26.20	75.0±10.4	169.7±8.6	26.04±2.8
5-9	58	11.09	78.0±6.2	170.4±10.6	26.89±1.7
10-14	49	9.37	77.5±6.8	168.5±6.8	27.29±3.0
15+	23	4.40	80.5±5.5	167.76±7.6	28.66±3.6

Table 3. Classification of infertile males according to period of infertility and BMI categories

Period of Infertility	No. of Males (523)	Classification									
		Under weight (<18.5)		Normal weight (18.5–22.9)		At risk of obesity (23-24.9)		Obese-I (25.0-29.9)		Obese-II (>30.0)	
		No	%	No	%	No	%	No	%	No	%
1-2	256	20	7.81	55	21.48	90	35.15	55	21.48	36	14.06
3-4	137	14	10.22	27	15.33	41	29.93	35	25.55	20	14.60
5-9	58	6	10.34	6	10.34	11	18.97	24	41.38	11	18.97
10-14	49	4	8.16	5	10.20	10	20.41	21	42.86	9	18.37
15+	23	2	8.70	2	8.70	4	17.39	7	30.43	8	34.78

Table 4. Distribution of the sample according to smoking and alcoholic habits

Type of Habit	No	%
Smoking	170	32.50
Alcoholism	125	23.90
Smoking & Alcoholism	205	39.19
Normal	23	4.39
Total	523	100.00

Distribution of male infertility cases by rating through percentage.



DISCUSSION

Male infertility is a multifactorial disease process with a number of potential contributing causes. Considering the majority of male infertility cases are due to deficient sperm production of unknown origin, environmental and nutritional factors must be evaluated. Lifestyle and dietary choices, Pesticide residues, and xenoestrogens all may adversely affect spermatogenesis [23]. Azoospermia is identified in approximately 1% of all men and 10% to 15% of infertile males [24]. With a population of approximately 3 billion people at reproductive age, a gross estimate indicates that approximately 10 million men worldwide are azoospermic. From the present study it is clear that the percentage of azoospermic males is (9.17%), these findings are in good agreement with other studies [25,26] and shows the regional variation

From this it is evident that the proportion of oligospermia affected males decreases (4.74%) as the period of infertility increases (15+ years). The findings from the other studies may suggest that abnormally low sperm counts adverse impact of factors in adulthood or as the result of a developmental problem. The evidence for declining sperm counts in recent decades mean that the environmental/lifestyle impact on spermatogenesis is an important health issue [27].

It is clear from the study that when the infertility period and BMI increases, the percentage of infertile individuals decreases. As a result of this, the present analysis indicates that there is a possible association with obesity and infertility. Age factor plays an important role in male infertility, the longer the period of infertility the more the number of obese individuals. In 15+ years of infertility period low percentage was observed (4.40%) with high BMI (28.66 ± 3.6). These findings are in good agreement with other studies that a retrospective analysis of data from 390 men suggested that high BMI was associated with reduced sperm concentration and motility [28].

It is observed from the present study that more percentages of males (39.19%) are infertile when they have both the habits of smoking and alcohol. A similar pattern

has been reported in other studies that Cigarette smoking has been association with adverse effects on fertility, although this is not widely recognized [29]. There is strong evidence of the adverse effects of smoking on fertility operating through a range of pathways in both the general and infertile population. In males, smoking negatively affects sperm production, motility and morphology and is associated with an increased risk of DNA damage [30,31].

Alcohol is a known teratogen [32,33] and its consumption has been reported to decrease fertility, although the level of consumption associated with risk is unclear. Alcohol consumption at the extreme level is known to be dangerous to the unborn child [34-36]. but the effect at lower levels is less certain. The mechanisms by which alcohol could impair conception are unclear but may include an alcohol-induced rise in estrogen, which reduces FSH secretion suppressing folliculogenesis and ovulation. It may also have a direct effect on the maturation of the ovum, ovulation, blastocyst development and implantation [37,38]. In contrast, in chronic alcoholics, there is good evidence for impairment of spermatogenesis and reductions in sperm counts and testosterone levels.

CONCLUSION

In conclusion male adiposity and lifestyle habits were associated with increased infertility in males. Increased adiposity could produce other biological changes in men that reduce their fertility. If such changes occur and are reversible, weight loss may improve their chances of conception. Lifestyle modification has the potential to improve reproductive performance raises a number of health-care issues. Most lifestyle factors are theoretically modifiable a structured programme of education, support and access to specialist health professionals should back counseling to encourage and facilitate appropriate lifestyle changes. This will facilitate the provision of optimum health care to couples attempting to become pregnant, improving their chances of success and minimizing the need for costly and invasive infertility treatment. The study is helpful to all the scientific, medical researchers who can put efforts to put end to male infertility.

REFERENCES

1. World Health Organization. (2010). WHO Laboratory manual for the examination and processing of human semen, 5th edition. Cambridge, Cambridge University Press.
2. Kumar D. (2007). Prevalence of female infertility and its socio-economic factors in tribal communities of Central India. *Rural and Remote Health*, 7, 456.
3. Dada R and Gupta N. (2004). CYqmicrodeletions Azoospermia factor candidate genes and spermatogenetic arrest. *Journal of Biomolecular Techniques*, 15, 176-183.
4. Peterson BD, Gold L, Feingold T. (2007). The experience and influence of infertility, considerations for couple counselors. *Fam J*, 15(3), 251-257.
5. Cooper TG, E Noonan, S Von Eckardstein et al. (2010). *Hum Reprod Update*, 16(3), 231-245.
6. Kulikauskas V, Blaustein D, Ablin RJ. (1985). Cigarette smoking and its possible effects on sperm. *Fertil Steril*, 44, 526-528.
7. De Celis R, Pedron-Nuevo N, Feria-Velasco A. (1996). Toxicology of male reproduction in animals and humans. *Arch Androl*, 37, 201-218.



8. Sharpe RM. (2000). Lifestyle and environmental contribution to male infertility. *British Medical bulletin*, 56, 630-642.
9. Marinelli D, Gaspari L, Pedotti P and Taioli E. (2004). Mini-review of studies on the effect of smoking and drinking habits on semen parameters. *Int J Hyg Environ Health*, 207, 185-192.
10. Martini AC, Molina RI, Estofan D, Senestrari D, Fiol de Cuneo M & Ruiz RD. (2004). Effects of alcohol and cigarette consumption on human seminal quality. *Fertil Steril*, 82, 374-377.
11. Villalta J, Balleca JL, Nicolas JM, Martinez de Osaba MJ, Antunez E & Pimentel C. (1997). Testicular function in asymptomatic chronic alcoholics, relation to ethanol intake. *Alcohol Clin Exp Res*, 21, 128-133.
12. Muthusami KR and Chinnaswamy P. (2005). Effect of chronic alcoholism on male fertility hormones and semen quality. *Fertil Steril*, 4, 19-924.
13. Must A, Spadano J, Coakley E, Field A, Colditz G, Dietz W. (1999). The disease burden associated with overweight and obesity. *JAMA*, 282, 1523-1559.
14. Catalano P. (2007). Management of obesity in pregnancy. *Obstet Gynecol*, 109, 419-433.
15. Jensen T, Andersson A, Jorgensen N, Andersen A, Carlsen E, Petersen J, Skakkebaek N. (2004). Body mass index in relation to semen quality and reproductive hormones among 1,558 Danish men. *Fertil Steril*, 82, 863-870.
16. Roudebush W, Witt M, Kort H, Massey J, Elsner C, Mitchell-Leaf D. (2005). Men with high body mass index values present with lower serum testosterone levels (abstract). *Fertil Steril*, 84, 179.
17. Fejes I, Koloszar S, Zavaczki Z, Daru J, Szollosi J, Pal A. (2006). Effect of body weight on testosterone/estradiol ratio in oligozoospermic patients. *Arch Androl*, 52, 97-102.
18. Magnusdottir E, Thorsteinsson T, Thorsteinsdottir S, Heimisdottir M, Olafsdottir K. (2005). Persistent organochlorines, sedentary occupation, obesity and human male subfertility. *Hum Reprod*, 20, 208-215.
19. Kort H, Massey J, Elsner C, Mitchell-Leaf D, Shapiro D, Witt M, Roudebush W. (2006). Impact of body mass index values on sperm quantity and quality. *J Androl*, 27, 450-452.
20. Weiner JS and Lourie JA. (1969). Human biology, A guide to field methods. IBP hand book No. 9, Black Well Scientific Publications, Oxford.
21. WHO. (1995). Physical status, Use and interpretation of Anthropometry. *WHO Tech Rep Ser*, 854.
22. Steering Committee. (2000). The Asia – Pacific perspective, Redefining Obesity and its treatment. Melbourne, International Diabetes Institute.
23. Steven Sinclair, ND, Lac, (2000). Male Infertility, Nutritional and Environmental Considerations. *Alternative Medicine Review*, 5(1), 28-38.
24. Cocuzza M, Alvarenga C, Pagani R. (2013). The epidemiology and etiology of azoospermia. *Clinics*, 68(S1), 15-26.
25. Patel Mital, Jain Shefali, Jain Dinesh, Patel Bhavesh, PhanseNandini, Vyas Priti and Rathore Pragya. (2012). Prevalence of Different Factors Responsible for Infertility. *Research Journal of Recent Sciences*, 1(ISC-2011), 207-211.
26. Rajvi H. Mehta, Sanjay Makwana, Geetha M, Ranga RJ, Srinivasan SS Virk. (2006). Prevalences of oligozoospermia and azoospermia in male partners of infertile couples from different parts of India *Asian J Androl*, 8(1), 89-93
27. Sharpe RM. (2010). Environmental/Life style effects on Spermato-genesis, *Phil Trans R Soc B*, 365, 1697-1712.
28. Hammoud AO, Gibson M, Petersen CM, Meikle AW & Carrell DT. (2008). Impact of male obesity on infertility, a critical review. *Fertil Steril*, 90, 897-904.
29. Roth LK and Taylor HS. (2001). Risks of smoking to reproductive health, assessment of women's knowledge. *Am J Obstet Gynecol*, 184, 934-939.
30. Zenzes MT, Bielecki R and Reed TE. (1999). Detection of benzo(a)pyrenediol epoxide-DNA adducts in sperm of men exposed to cigarette smoke. *Fertil Steril*, 72, 330-335.
31. Kunzle R, Mueller MD, Hanggi W, Birkhauser MH, Drescher H and Bersinger NA. (2003). Semen quality of male smokers and nonsmokers in infertile couples. *Fertil Steril*, 79, 287-291.
32. Randall CL. (1987). Alcohol as a teratogen, a decade of research in review. *Alcohol*, 1, 125-132.
33. Chaudhuri JD. (2000). An analysis of the teratogenic effects that could possibly be due to alcohol consumption by pregnant mothers. *Indian J Med Sci*, 54, 425-431.
34. Astley SJ, Bailey D, Talbot C and Clarren SK. (2000). Fetal alcohol syndrome (FAS) primary prevention through fas diagnosis, II. A comprehensive profile of 80 birth mothers of children with FAS. *Alcohol Alcohol*, 35, 509-519.
35. Goransson M, Magnusson A, Bergman H, Rydberg U and Heilig M. (2003). Fetus at risk, prevalence of alcohol consumption during pregnancy estimated with a simple levels and reproductive function. Ie screening method in Swedish antenatal clinics. *Addiction*, 98, 1513-1520.
36. Krulewitch CJ. (2005). Alcohol consumption during pregnancy. *Annu Rev Nurs Res*, 23,101-134.
37. Eggert J, Theobald H and Engfeldt P. (2004). Effects of alcohol consumption on female fertility during an 18-year period. *Fertil Steril*, 81, 379-383.
38. Gill J. (2000). The effects of moderate alcohol consumption on female hormone levels and reproductive function. *Alcohol Alcohol*, 35, 417-423.

