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RESEARCH ARTICLE

Does Weight Loss improve Fertility with respect to Semen Parameters—Results from a Large Cohort Study

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ABSTRACT

Objective: To determine whether weight loss in obese men improves seminal parameters.

Design: Prospective interventional study.

Setting: Infertility clinic and weight loss centers.

Patient: All men attending infertility center and weight loss programmes from April 2012 to May 2015 (n = 105).

Intervention: Diet counseling and exercise.

Main Outcome Measure: Collected reproductive parameters included semen analysis (ejaculate volume, sperm concentration, progressive and non-progressive motility) data. Body mass index (BMI) was calculated for all patients with comparisons to reproductive parameters before and after weight loss by using paired-t test and *Chi-square* tests.

Result: The mean BMI was significantly higher before weight loss (33.2) than after weight loss (30.4) in obese men. The weight loss had significant positive correlation with percentage of progressive sperm motility (p = < 0.001) and static percentage (p = < 0.001). Weight loss had non-significant correlation with semen volume (p = 0.083), concentration (p = 0.418) and non progressive motile sperm (p = 0.361).

Conclusion: In one of the largest cohorts of male fertility and obesity, semen parameters demonstrated mild but significant relationships with BMI and semen parameters, possibly contributing to subfertility in this population.

Keywords: Body mass index, Male infertility, Obesity, Semen analysis, Weight loss.

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INTRODUCTION

According to the World Health Organization (WHO), infertility is "a disease of the reproductive system defined by the failure to achieve a clinical pregnancy after 12 months or more of regular unprotected sexual intercourse." Most of the developed countries are bearing the brunt of high frequency of overweight or obesity.[1] In the developing world, the prevalence of overweight and obesity is increasing at alarming rates,[2-6] and in some countries it has reached levels observed in the developed world.[7,8] Although overweight and obesity have been demonstrated to affect female fertility,[9] there is no consensus on the effects on male fertility and seminal parameters.[10-12] Recent epidemiological studies conducted on male infertile couples suggest that obesity is associated with decline in male fertility.[13] Male obesity has been linked to subfecundity and an effect–response relationship between increasing body mass index (BMI) and subfecundity has been proposed.[14-16] Urbanization is one of the main reasons for higher prevalence of obesity in developing countries in terms of food intake and physical activity. Apart from poor lifestyle, genetic makeup also plays a role in increasing obesity in India. India and most of the Asia-Pacific population have mutation of MC4R genes, which puts them into high-risk population.[17] The WHO has reclassified the obesity limits for Asians/Indians as indicated in Table 1.[18]

It is well documented that there is a strong association between obesity and poor semen parameters,[19-26] although with conflicting results.[11,27-32] Obesity has shown to affect the gonadotropin-releasing hormone–luteinizing hormone/follicle-stimulating hormone (GnRH-LH/FSH) pulse that may impair Leydig and Sertoli cell functions and with the release of sex hormones with consequent effect on sperm maturation.[33] It is demonstrated that

Table 1: The WHO-recommended classification for Indian population

Body mass index cut-off values for adults		
		Asians/Indians 2000
Underweight	<18.5	<18.5
Normal range	18.5–24.9	18.5–22.9
Overweight (preobese)	25.0–29.9	23.0–24.9
Obese class I	30.0–34.9	25.0–29.9
Obese class II	35.0–39.9	≥30.0
Obese class III	≥40.0	

1 WHO[8] /Technical Report Series (TRS 894)

2 WHO/The International Obesity Task Force (IOTF)/International Association for the Study of Obesity (IASO) [18]

altered hormonal profile in obese men have negative impact on semen parameters.

Decreased levels of gonadotropins, free testosterone, sex hormone-binding globulin and also increased levels of estrogens are mainly associated with obesity-related infertility.[34-37] All these alterations might affect the male reproductive system and gamete quality. Previous studies have shown that the endocrine abnormalities may be reversed by weight reduction.[38-43] Our study aimed to find the changes in different semen parameters before and after weight loss program in obese men.

MATERIALS AND METHODS

Obese men aged 25 to 40 years (mean age = 32.5 ± 7.5) with BMI more than 25 kg/m^2 were recruited for the study. All the participants were subjected to weight loss intervention and followed up for 1 year after weight loss.

We excluded two men diagnosed with Klinefelter's syndrome, and in the analyses of semen characteristics, two with retractile testis and seven men with azoospermia were excluded because azoospermia probably is not caused by obesity alone. The weight loss program was based on healthy diet and exercise that lasted for approximately 3 months. All the participants were asked to fill up the questionnaires related to their reproductive activities, medical and surgical history, and their lifestyle before and after weight loss intervention.

Prader orchidometer was used to measure the volume of each testis. The clinical examination was performed on site by one investigator and included blood pressure by mercurial sphygmomanometer, blood sugar level (Accucheck), and height and weight measurements (Table 2).

A sterile nontoxic container was provided to the participants to collect the semen sample by masturbation. They were instructed to maintain at least 48 hours of abstinence. Participants were given the option of semen collection at home on their request. A written consent was taken from the participants and no incentives were given for the participation. The study was approved by our regional ethics committee with the approval number as P-RM-08/2012.

Table 2: Characteristics of study participants

Parameter	*Before intervention*	*After intervention*	*% change*
BMI <18.5 (kg/m²)	0 (0%)	0 (0%)	0.0%
BMI 18.5–24.9 (kg/m²)	0 (0%)	11 (10.5%)	10.5%
BMI 25.0–29.9 (kg/m²)	25 (23.8%)	47 (44.8%)	21.0%
BMI 30.0–34.9 (kg/m²)	57 (54.3%)	31 (29.5%)	–24.8%
BMI 35.0–39.9 (kg/m²)	12 (11.4%)	7 (6.7%)	–4.7%
BMI ≥40.0 (kg/m²)	11 (10.5%)	9 (8.6%)	–1.9%
Smoking	34 (32.4%)	34 (32.4%)	0.0%
Alcohol consumption	21 (20%)	14 (13.3%)	–6.7%
Hypertension	10 (9.5%)	11 (10.5%)	1.0%
Diabetes	7 (6.7%)	7 (6.7%)	0.0%

BMI: Body mass index

ANALYSES OF SEMEN SAMPLES

Semen volume was estimated by 3 mL Pasteur pipette (Tarsons). Sperm concentration and motility were assessed as described in "WHO Laboratory Manual".[8] Semen sample analysis was performed within 1 hour after ejaculation, since sperm motility is stable within this time.[44] CASA system (CASA, Nikon, Model Eclipse E200) was used for count and motility. Mackler chamber was also used to countercheck the concentration.

Men diagnosed with azoospermia or severe oligospermia (<5 × 10^6) were ruled out for Y-chromosome microdeletions. Then they were subjected to weight reduction program, which includes diet counseling, exercise, and/or lipolysis for a duration of minimum 3 months. All the previous tests were repeated once again and the results were compared with their previous results and the difference studied.

Statistical Methods

Descriptive and inferential statistical analysis has been carried out in the present study.[45-48] Continuous variables were presented as mean ± standard deviation. Categorical variables were presented as percentages and number of events. We used paired-*t* test and *Chi-square* tests to compare the means and evaluate their associations. Data were collected on Microsoft Excel on a standardized sheet. Analysis was performed using Statistical Package for the Social Sciences version 12.0. P-value of <0.05 was regarded as significant.

RESULTS

Data were prospectively collected between April 2012 and May 2015. A total of 213 men were invited to participate of which 126 men (57%) were recruited. Out of the 126 participants, 21 were excluded according to our exclusion criteria (16%), and remaining 105 were actually recruited for our weight loss program.

The data of the studied groups are represented in Table 3. The mean BMI was significantly higher in group I (before weight loss) than in group II (after weight loss) obese men. We observed that weight loss improved the percentage of sperm motility, while the percentage of static sperm decreased. Weight loss had nonsignificant correlation with sperm concentration, nonprogressive sperm percentage, and a suggestive significant correlation with semen volume (Table 3 and Graphs 1A to F).

Study design: Prospective interventional study

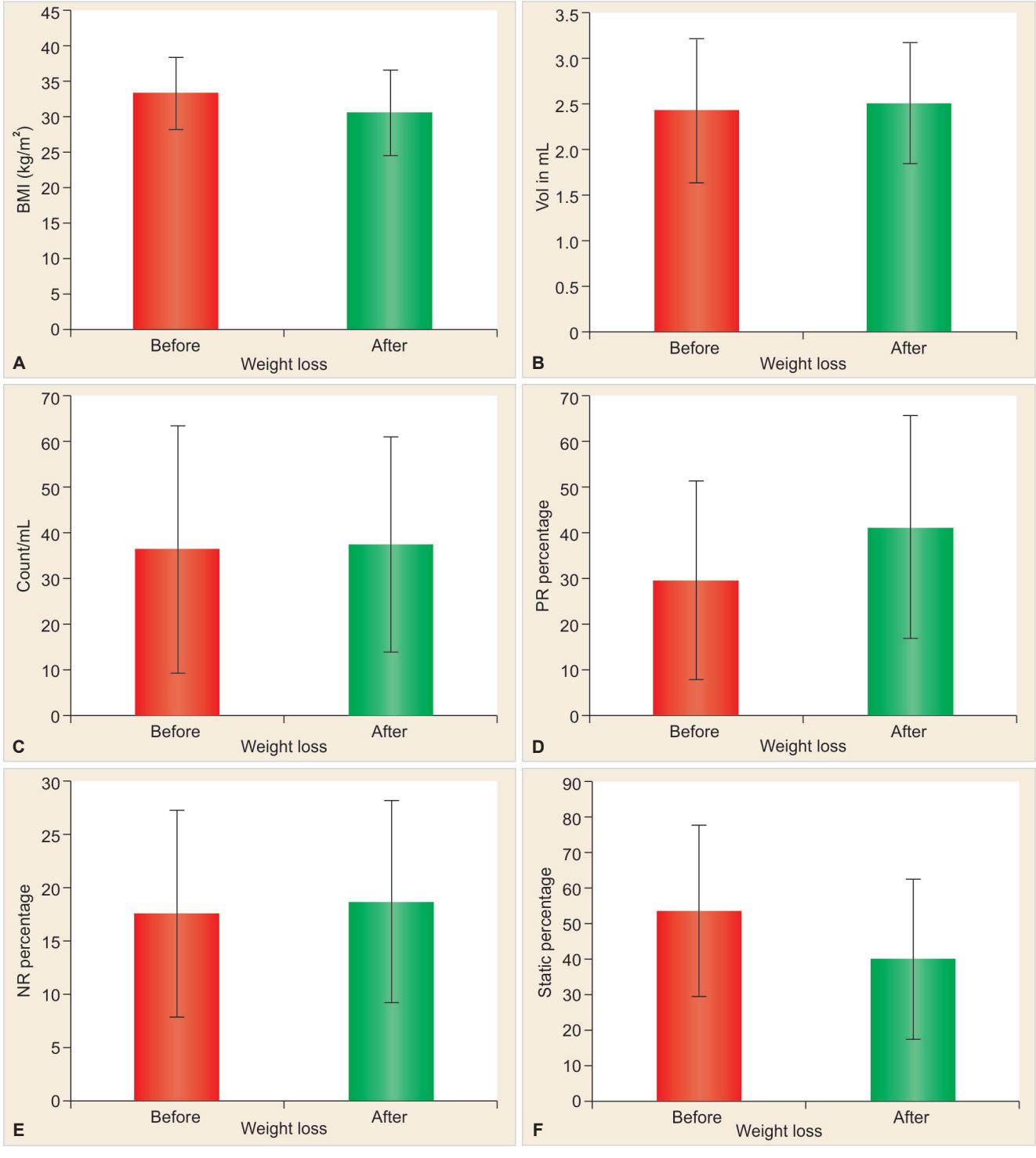
DISCUSSION

Studies are limited regarding the impact of weight loss with diet and exercise on male fertility. Most of the

Table 3: Assessment of study variables before and after intervention

Variables	*Before intervention*	*After intervention*	*Difference*	*t-value*	*p-value*
Body mass index (kg/m^2)	33.18 ± 5.06	30.43 ± 5.98	2.755	10.257	<0.001**
Vol in mL	2.44 ± 0.80	2.52 ± 0.67	–0.085	–1.751	0.083†
Count/mL	36.38 ± 27.05	37.43 ± 23.46	–1.051	–0.814	0.418
Progressive % (PR)	29.35 ± 21.91	41.06 ± 24.40	–11.712	–4.282	<0.001**
Non-progressive % (NR)	17.57 ± 9.71	18.70 ± 9.49	–1.130	–0.917	0.361
Static %	53.70 ± 24.27	39.97 ± 22.50	13.737	5.386	<0.001**

Student's t test (paired); †suggestive significance ($0.05 < p < 0.10$); moderately significant ($0.01 < p \leq 0.05$); **strongly significant ($p \leq 0.01$)



Graphs 1A to F: Impact of weight loss on different seminal parameters

available studies are conducted in clinical setup, where the participants tend to be infertile. This study evaluates the effects of obesity and weight loss interventions in male fertility in the general population.

Our study showed that a high BMI was associated with low values of sperm concentration, total sperm count, and percentage of motile sperm.

We did not observe statistically significant differences in sperm concentration, volume, or nonprogressive sperm count. Weight loss was associated with an increase in the percentage of progressive motile sperm and decrease in static sperm percentage among men who participated in a 12-week weight loss program. Our results indicate that there is a causal inverse association between BMI and semen quality, suggesting that it may be possible to improve semen quality by weight reduction. Previous studies have reported a higher prevalence of oligospermia in overweight and obese men than in normal weight men, with a significant association between sperm count and BMI.[19]

A study conducted on normal weight and obese men, both having normal semen parameters, reported lower sperm count in obese group compared with the normal group.[49] It is well established that obesity also has a negative impact on the morphology of sperm.[23]

Obesity has been implicated to have direct and indirect impacts that could reduce male fertility including decreased sperm motility and increased DNA fragmentation.[14,16] Obese men suffering from sleep apnea have shown improved testosterone levels after weight loss.[50] There is a strong correlation between physical activity and erectile function. Physical activity has been shown to improve sexual activities, especially among males who were less active earlier.[51] Obese men with metabolic syndrome also have shown improvement in erection function after reducing caloric intake.

Improvement in sexual activity is also reported after weight loss by gastric bypass surgical means.[52] Even though natural weight loss and gastric bypass have shown promising results in terms of restoring fertility and surgical treatment has been shown to restore reproductive hormones to normal levels, some studies indicate that gastric bypass procedure and the drastic weight loss might induce secondary infertility. According to a report, six morbidly fertile obese men lost 60 to 80 kg after a Roux-en-Y gastric bypass developed secondary infertility and complete azoospermia and ultimately landed up with complete spermatogenic arrest.[53] The BMI does not directly reflect the percentage of body fat, but is a measure of weight in relation to height. Some muscular men may have high BMI despite not being obese, which may not be associated with poor semen quality.

The BMI does not directly reflect the percentage of fat as such, but is a measure of weight in relation to height. Few men have high BMI because of large muscle mass, such men may not have compromised semen quality. Chances of influence on our findings by different confounders like smoking, alcohol, diabetes mellitus, hypertension, and abstinence time are obvious. We did, however, take into account these factors in the regression analyses and they are unlikely to explain our findings.

It should be noted that few men in spite of losing weight remained overweight/obese after the weight loss program.

CONCLUSION

To conclude on this study, we observed that the altered androgen profile tended to improve following weight loss and that weight loss may potentially lead to improvement in semen quality, although we cannot conclude this to be a result of the reduction in body weight *per se*.

A lot of research still needs to be carried out to clear the gray area to set up concrete mechanisms involved in obesity and develop an effective treatment protocol for obese infertile men. There is enough clinical evidence regarding obesity and its role in infertility, so there should be a different approach implemented toward preconception management in obese men. However, the available clinical evidence is strong enough for health policies to be implemented immediately in public and private settings regarding preconception counseling and different approach of treatment for obese population without further delay. The promotion of weight excess prevention and control, especially through lifestyle therapies, should be mandatory not only for improving reproductive and obstructive outcomes but also for reducing the cost of medical treatment eradicating or treating the secondary diseases with obesity as the root cause.

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