Original Article

Relationship between body mass status and semen quality

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ABSTRACT

Obesity is on a steep rise even in developing countries. Both male and female factors can contribute to hypoactive reproductive functions. Obesity as a contributing factor to failure of reproductive functions in women is an established fact. But literature available on obesity in males on reproductive function failure is very minimal. Body Mass Index (BMI), Waist Circumference (WC), Hip Circumference (HC) and Waist Hip Ratio (W/H ratio) are frequently used to evaluate body mass status. Methods: 300 normal healthy male volunteers in the age group 20-35 years selected randomly. The BMI was calculated, WC and HC measured and semen analysis done as per WHO guidelines. Results: Statistical analysis was done using SPSS 17. Semen volume, sperm count, sperm motility and morphologically normal sperms showed a negative correlation with all anthropometric measures. Conclusion: A combination of factors like increased estrogen & decreased androgen levels in circulation, hormone leptin secreted by adipocytes modulating and decreasing testicular activity, inflammation by adipocytokines damaging the germ cells and adipose tissue deposits in the testes increasing the testicular temperatures may be attributed as a cause for decrease in reproductive function in obese males. The multi pronged attack by obesity on reproductive function makes it one of the most important factors to be considered for reproductive hypoactivity in men. But fortunately, since, obesity can be reversed by proper corrective measures like diet, exercising or even bariatric surgery in morbidly obese, the hypoactive reproductive functions can be dealt with successfully.

1. Introduction

Obesity is found to be on the rise not only in developed countries but also in developing countries. Obesity has created a lot of health problems involving various systems of the body, including the reproductive system, for both women and men. In our society, the blame for infertility is usually attributed to females, though, in many cases the contribution is from the male counterpart. The cause of decreased reproductive function may be either due to female factors or male factors or combination of male and female factors. Body Mass Index (BMI), Waist Circumference (WC), Hip Circumference (HC) and Waist/Hip ratio (W/H ratio) are frequently used to identify obese individuals. Many studies have shown that the pattern of fat distribution in the body to have a positive correlation with abnormalities than fat distribution by itself. Obesity in women and its relation to reproductive and fertility problems such as PCOS, birth defects, spontaneous abortions and miscarriages is well studied [1-4]. But literature for the effect of obesity on male reproductive function is minimal. So this study was undertaken to find the correlation, if any, exist between body mass status & semen parameters. Since the site of deposition of fat has an important effect on health our objective was to correlate these various parameters and find out if there exists a link between certain obesity indices such as BMI, WC and HC with semen quality (semen volume, sperm count, sperm motility and sperm morphology). The basic and most widely used test to assess the male fertility is still the standard semen analysis.
The present study was conducted in a tertiary care hospital after acquiring the approval of the ethics committee and of the head of the institution. It involved 300 normal healthy male volunteers in the age group of 20-35 years selected randomly. The procedure was explained to the subjects and informed consent was taken to participate in the study. Any subject with past and/or present history of medical treatment for any testicular pathology or Diabetes Mellitus or Tuberculosis or Leprosy or who had Mumps infection or who had any surgical procedure of genital tract were excluded from the study. An appointment for semen analysis was given to the subjects. Instructions regarding abstinence from sexual intercourse as well as masturbation for a period of 3-5 days prior to the appointment date were given to subjects. Subjects were called in the morning at 9.00 am on the appointment date at the semen analysis laboratory. The height was recorded using a stadiometer and the weight measured using a scientific weighing scale. The BMI was calculated by Quetlet's index \[\text{BMI} = \frac{\text{weight (in kgs)}}{\text{height (in metres)}^2}\], which is the weight to height ratio and used to identify overall obesity. The waist circumference was measured at the end of normal expiration using a measuring tape measured halfway between iliac crest and the bottom of 12th costal bone. Hip circumference was measured at the level of tuberculli majoris using the same measuring tape. The waist and hip circumference indicate central or abdominal obesity. The semen analysis was carried out after the liquefaction and as given below as per the guidelines laid by World Health Organization [6].

Volume of the semen was measured in a graduated glass cylinder with a conical base. A drop of semen was placed on a slide and microscopic examination was done under 400X magnification to determine the sperm aggregation, the evenness of spread of spermatozoa on the slide and also to determine the dilution required for hemocytometry evaluation of total count. At least 5 microscopic fields were assessed in a systematic way to classify 200 spermatozoa. The motility of the sperms were determined and noted. Sperm morphology was evaluated by staining with Papanicolaou stain according to WHO guidelines. The smear was observed using 100X oil immersion objective lens. At least 200 sperms were examined for characteristics of normal spermatozoa as per WHO guidelines. Sperms with defects in head, mid piece or tail were taken as abnormal spermatozoa. The percentage of morphologically normal sperms was calculated. Percentage of morphologically normal forms more than 35% considered within normal limits [6].

### 2. Materials and Methods

The statistical analysis was done using SPSS 17. The mean and SD were calculated and correlation among various factors done using Sig 2 tailed Pearson’s correlation. The subjects in this study were of mean age 25.76± 3.16, weight 70.21 ± 10.20 kgs, height 167.21 ± 6.81 cms, BMI 25.08 ± 3.097, Waist circumference 82.34 ±12.07 cms, HC 88.91 ±10.73 cms (Table 1) The correlation between each of the anthropometric measurement (BMI, WC & HC) with semen parameters were found out individually using Pearson’s correlation formula. The results showed BMI, WC and HC to have a highly significant negative correlation with semen volume, sperm count and motility of sperms. BMI and HC showed a highly significant correlation with morphologically normal sperms whereas WC showed a significant correlation (Table 2).

### Table 1. Distribution of characteristics of study population

<table>
<thead>
<tr>
<th></th>
<th>Age in Years</th>
<th>Weight (kg)</th>
<th>Height (m)</th>
<th>BMI</th>
<th>WC (cms)</th>
<th>HC (cms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>25.76</td>
<td>70.21</td>
<td>167.2</td>
<td>25.08</td>
<td>82.34</td>
<td>88.91</td>
</tr>
<tr>
<td>Std. dev</td>
<td>3.16</td>
<td>10.2</td>
<td>16.81</td>
<td>3.09</td>
<td>12.07</td>
<td>10.73</td>
</tr>
</tbody>
</table>

### Table 2 Correlation of BMI, WC & HC with semen volume, sperm count, sperm motility & morphologically normal sperms

<table>
<thead>
<tr>
<th></th>
<th>BMI</th>
<th>WC</th>
<th>HC</th>
<th>SEMEN VOLUME</th>
<th>SPERM COUNT</th>
<th>MOTILE SPERM</th>
<th>MORPHOLOGICALLY NORMAL SPERM</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI Pearson Correlation</td>
<td>1</td>
<td>.787**(*)</td>
<td>.758**(*)</td>
<td>-.314**(*)</td>
<td>-.547**(*)</td>
<td>-.555**(*)</td>
<td>-.555**(*)</td>
</tr>
<tr>
<td>Sig (2-tailed)</td>
<td>.000</td>
<td>.000</td>
<td>.000</td>
<td>.000</td>
<td>.000</td>
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<td>N</td>
<td>300</td>
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<td>300</td>
<td>300</td>
<td>300</td>
<td>300</td>
</tr>
<tr>
<td>WC Pearson Correlation</td>
<td>.787**(*)</td>
<td>1</td>
<td>.937**(*)</td>
<td>-.274**(*)</td>
<td>-.631**(*)</td>
<td>-.616**(*)</td>
<td>-.616**(*)</td>
</tr>
<tr>
<td>Sig (2-tailed)</td>
<td>.000</td>
<td>300</td>
<td>.000</td>
<td>.000</td>
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<tr>
<td>N</td>
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<td>300</td>
<td>300</td>
<td>300</td>
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<td>300</td>
</tr>
<tr>
<td>HC Pearson Correlation</td>
<td>.758**(*)</td>
<td>.937**(*)</td>
<td>1+</td>
<td>-.266**(*)</td>
<td>-.561**(*)</td>
<td>-.544**(*)</td>
<td>-.544**(*)</td>
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<tr>
<td>Sig (2-tailed)</td>
<td>.000</td>
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<td>000</td>
<td>.000</td>
<td>.000</td>
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<td>.002</td>
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</tbody>
</table>

** Correlation is significant at the 0.01 level (2-tailed).
* Correlation is significant at the 0.05 level (2-tailed).
4. Discussion

The semen samples were collected from 300 randomly selected normal healthy male volunteers in the age group 20-35 years and were evaluated for volume, count, motility, and morphology. The semen parameters were correlated with BMI, WC and HC. All aspects of semen parameters show a negative correlation with BMI, HC and WC and thereby show the detrimental effect of increasing body mass on sperm parameters irrespective of whether it is upper body or central obesity.

The synthesis of Testosterone and gametogenesis by the testes is controlled by hypothalamic-pituitary-testicular (HPT) axis. Testicular injuries, diseases, impairments of the HPT axis or obesity can produce abnormalities of spermatogenesis resulting in production of fewer, abnormal or underdeveloped sperms with morphological defects and reduced motility.

Increase in adipocytes or fat cells in the body results in obesity. The adipocytes in their intercellular septum have Cytochrome p450, an aromatase enzyme, which plays an important role in regulation of triglycerides and aromatization of androgens to estrogens. Estrogen is a positive regulator and androgens a negative regulator of adipogenesis [7]. Overweight subjects hence show more estrogen production [8-15] and a decrease in Inhibin B, the marker of spermatogenesis [13-15].

Adipocytokines synthesized by adipocytes causes inflammation and the release of free radicals which causes damage to the sperms and reduces their concentration and motility. Adipocytokines also secrete hormone Leptin whose circulating level is directly proportional to the total amount of fat in the body [9, 16, 17]. Leptin acts on hypothalamic satiety centre and decreases the weight. But hypothalamic resistance to Leptin increases the body weight and fat levels and Leptin also modulates testicular physiology and causes decreased testicular function resulting in decreased testosterone production. [8, 9] This also causes a decrease in androgen dependent Neutral alpha glucosidase (NAG) enzyme secreted in the epididymal fluid which is necessary for epididymal maturation of sperms, whereby the sperms acquire motility [18].

The deposition of adipose tissue around the pampiniform plexus causes testicular heat stress in animals [9,19]. Testicular heat-stress responses along with associated apoptosis, gene expression and inhibition of DNA repair, results in apoptosis and altered spermatogenesis [9,20].

5. Conclusion

Hence it can be speculated that a combination of factors cause the decrease in reproductive function in obese males. This includes 1) aromatization of testosterone to estrogen by adipocytes 2) thereby causing an increased estrogen & decreased androgen levels in circulation 3) inflammation caused by adipocytokines damaging the germ cells 4) Decreased enzyme Neutral Alpha Glucosidase (NAG) in the epididymal fluid which is necessary for the sperms to acquire motility in epididymis 5) Hormone leptin secreted by adipocytes fails to bring about satiety in cases of hypothalamic resistance to Leptin and also causes a decreased testicular activity 6) adipose tissue deposits in the testes increases the testicular temperatures, thereby triggering apoptosis pathways. The multi pronged attack of obesity on reproductive function makes it one of the most important factors to be considered for reproductive hypoaactivity in men. This study does not ascertain the body mass status at which the reproductive functions are affected and whether the reproductive functions are reversible with reversal of obesity. This requires further work in this area.

Fortunately, since, obesity can be reversed by proper corrective measures like diet, exercising or even bariatric surgery in morbidly obese, hypopactive reproductive functions can be dealt with successfully. Greater awareness of the effects of obesity on fertility, better understanding of causative mechanisms and effective efforts to limit or eradicate obesity has to be ensured from public health angle.

6. References


