Association of Obesity with Male Infertility among Infertile Couples is not Significant in Mysore, South India

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Abstract

Objectives: In the present study we made an attempt to assess the spermiogram of the overweight and obese males of infertile couples seeking fertility treatment compared with healthy non-obese fertile individuals. Materials and Methods: A total of 404 subjects were recruited for this study from Mysore, India. BMI was measured for both cases and control subjects using existing cut off points in India and participants were divided into three categories including normal BMI, overweight and obese males. Semen samples were collected from the participants after 3-5 days of ejaculatory abstinence and were analyzed according to WHO guidelines. All tests performed in duplicates and mean values of the same were recorded and worked out for statistical analysis using SPSS 16.0 version. Results: No significant association between obesity in infertile male among infertile couples was observed. Conclusion: Although our data did not a significant incidence of male obesity associated with couple’s infertility
but it is still suggested to decrease weight to prevent hormone imbalance which is a reason for male subfertility.

**Keywords:** Obesity; BMI; male infertility; spermiogram.

1. **Introduction**

Infertility is the inability of a sexually active, non-contracepting couple to achieve pregnancy in one year.\(^1\) It is estimated that globally, 60–80 million couples suffer from infertility every year, of which probably 15–20 million cases are in India alone.\(^2\) Male causes for infertility are found in 50% of couples who undergo diagnostic tests to elucidate the underlying causative factor.\(^3\) The etiology of the male factor infertility is multifactorial and these affect spermatogenesis, which is a complex cascade of events governed by several genes and hormones. Obesity is rapidly increasing worldwide.\(^4,5\) Excess weight is not only linked to increased risk of chronic disease, \(^6\) but has also been reported to increase the risk of reproductive problems.\(^7\) Body fatness is most commonly assessed by Body Mass Index (BMI) which is calculated by dividing an individual’s weight measured in kilogram’s by their height in meters squared.\(^24\) Total body fat, intra abdominal fat, and subcutaneous fat have all been associated with low levels of total and free testosterone.\(^23\) Obese men have been reported to exhibit higher levels of circulating estradiol.\(^3\) Excess body fat also impairs production of the gonadotropin releasing hormone (GnRH).\(^19\) Over recent decade, several authors have published that overweight and obesity are considered factors induce a decrease sperm counts. It has also been estimated that sperm counts have been decreasing by as much as 1.5% each year in the United States.\(^6,21\) However, there is controversy regarding the extent of the relationship between obesity and male infertility and its mechanisms. Jensen et al, [2004] reported a higher prevalence of oligozoospermia in overweight and obese men compared with normal-weight men.\(^15\) In a group of 274 men, obesity was also associated with a lower sperm count compared with non-obese males. This relationship was only reported in normozoospermic men.\(^9\) Kort et al.\(^8\) found that BMI correlated negatively with the total number of normal spermatozoa. They did not report on sperm count or morphology. Furthermore, a study in India found a strong association of increased BMI on impaired semen parameters among obese males.\(^13\) To answer all these contradicting reports the present study was aimed to assess the extent of the relationship between male infertility and BMI among couple infertility and its mechanisms by studying the semen parameters and BMI in a large number of male partners of infertile couples.
2. Materials and Methods

The present study was conducted in Mediwave IVF and Fertility Research Hospital, Mysore, Karnataka, India, over a period of 2 years, starting from January 2010 to January 2012. A total of 352 infertile couples who approached the Mediwave IVF and fertility research hospital, for the inability to conceive were recruited in this study. Further, 52 potent males who were naturally conceived or represented two consecutive normal semen parameters were added as controls. The male partners of infertile couples were checked for obesity and infertility. The study was approved by the institutional ethical clearance committee of the University of Mysore, concerned hospital and IVF center. Informed written consent letter was obtained from the participants before including them in this study. All male partners were interviewed to collect information about family history, medical history, reproductive history and lifestyle factors which includes age, the duration of active marriage life, duration of infertility, sexual relationship, frequency of coitus, premature ejaculation, psychological status and libido was noted. Infertile males with anatomical, genetical, infection (past history of infection like mumps, measles, pneumonia, sexually transmitted diseases (STD), tuberculosis, environmentally induced (occupational exposure to radiation, heat) any surgical procedure and etiologies were excluded from the study. Habits like alcohol consumption, smoking and other substance abuse was also recorded.

2.1. Semen collection and analysis
After 3-5 days of ejaculatory abstinence the semen samples were collected in the laboratory room in a clean, dry, biologically inert container by the process of masturbation. In case of oligospermic or azoospermic patients, three semen samples were collected on alternate day and thorough examination was carried out. The collected samples were allowed to liquefy at 37°C for 30 minutes and analyzed within one hour after collection. Physical examination such as liquefaction time, odor, color, pH and viscosity were recorded after liquefaction. Microscopic examinations were carried out to record the semen volume, count, total count, motility and morphology of the sperm according to WHO criteria (1999).

2.2. Body Mass Index Calculation
Height (m) and weight (kg) were recorded on the day of semen collection for both cases and controls. BMI was calculated based on WHO growth charts and cutoff points and expressed as kg/m². Patients were grouped according to published BMI ranges.

2.3. Statistical analysis
To estimate the levels of different sperm parameters for both study groups, mean and standard deviation of the same were worked out. To compare the same, t-test was
performed. Data was entered into the computer using Microsoft Excel and analyzed using SPSS 16.0 version.

3. Results and discussion

In the present study, among 352 infertile couples, 36 males found with BMI <20 kg/m$^2$, 180 males with BMI 20-25 kg/m$^2$, 139 males with BMI 25-30 kg/m$^2$ and 49 males were found with BMI>30 kg/m$^2$ (Table 1). For semen parameters, 73 male partners of the infertile couples passed all the criteria and were considered as noromozoospermic. Further, 279 males were found with abnormal semen parameters and divided into different sub-groups according to the seminal condition (Table 2). The data on the infertility conditions among male partners of infertile couples has been depicted in Table 2.

Obesity is a major health issue and the relationship between obesity and male infertility has been described recently in many reports.$^{[11-13, 20]}$ Also, men with high BMIs typically are found to have an abnormal semen analysis as well. Jensen et al. reported a higher prevalence of oligozoospermia in overweight and obese men compared with normal BMI.$^{[15]}$ However, they did not find any relationship between increasing male BMI and percentage of motile sperm. Kort et al. $^{[8]}$ found that BMI correlated negatively with the total number of normal spermatozoa. They did not report on sperm count or morphology. In a recent study in India, the negative correlation was found between male BMI and sperm parameters like sperm count and motility $^{[9]}$ and stated that obesity may lead to male infertility by increasing lipid peroxidation.$^{[17]}$ In a trial to improve the semen quality it was reported that; reproductive hormone levels have been shown to normalize after weight loss.$^{[10,22]}$ It remains, however, to be seen whether weight loss may also improve semen quality. Sallmen et al. $^{[19]}$ stated that, hormone irregularities in men affect stimulation of the testicles that inhibit sperm production.$^{[14]}$ Several studies have reported reductions in testosterone with obesity.$^{[15,16,21]}$ In massively obese individuals, reduced spermatogenesis associated with severe hypotestosteronemia may favor infertility. $^{[19,23]}$ In another study, overweight and obese men had reduced sperm motility and increased sperm DNA fragmentation.$^{[8]}$ Contrary to other recent studies, no increased risk was observed in the present study among obese men, could be due to the less sample size. Many studies lacked information on frequency of sexual intercourse so obesity-related changes in sexual function could not be distinguished from obesity-related effects on fertility.$^{[19]}$ Because obesity has been associated with both sexual and erectile dysfunction $^{[24,25]}$, therefore reduced intercourse frequency could be a mediating factor by which obesity produces infertility. In our study we examined the relationship between frequency of intercourse and men’s BMI. But the association was negative thus; the mechanism that explains the BMI effect is likely to involve hormones rather than semen changes or sexual function. All previous studies mainly
focused on the effect of obesity on male infertility but none of them assessed the incidence of obesity among infertile couples and as per our knowledge this study is the first in India which analyzed the prevalence of overweight and obesity in infertile couples.

4. Conclusion

In conclusion, although our data found no significant relation between increased BMI in males and negative changes in their semen parameters in infertile couples, but it is still suggested to reduce weight in obese males to prevent hormone imbalance which may indirectly lead to sub-fertility.

References

**Table 1.** BMI and semen parameters among males of infertile couples and control males excluding azoospermia and aspermia (n=289).

<table>
<thead>
<tr>
<th>BMI</th>
<th>No. of Subjects</th>
<th>Volume</th>
<th>Count</th>
<th>Total Count</th>
<th>Motility</th>
<th>Vitality</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20</td>
<td>Normal-14</td>
<td>2.07±0.77</td>
<td>64.64±24.33</td>
<td>133.03±71.18</td>
<td>56.14±11.31</td>
<td>70.21±11.57</td>
</tr>
<tr>
<td></td>
<td>Infertile-8</td>
<td>1.13±0.67</td>
<td>25.75±32.89</td>
<td>15.16±18.03</td>
<td>17.5±11.63</td>
<td>48.5±23.77</td>
</tr>
<tr>
<td>&gt;20-25</td>
<td>Normal-54</td>
<td>2.03±0.61</td>
<td>59.65±21.18</td>
<td>120.93±58.88</td>
<td>56±12.76</td>
<td>73.75±10.54</td>
</tr>
<tr>
<td></td>
<td>Infertile-69</td>
<td>1.69±0.79</td>
<td>15.78±17.9</td>
<td>23.18±24.43</td>
<td>15.34±15.01</td>
<td>47.33±22.16</td>
</tr>
<tr>
<td>&gt;25-30</td>
<td>Normal-47</td>
<td>1.95±0.75</td>
<td>60.61±22.56</td>
<td>116.53±65.82</td>
<td>53.38±10.78</td>
<td>71.87±10.85</td>
</tr>
<tr>
<td></td>
<td>Infertile-63</td>
<td>1.62±0.75</td>
<td>16.28±16.16</td>
<td>23.91±24.64</td>
<td>18.74±17.33</td>
<td>49.85±20.94</td>
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<tr>
<td>&gt;30</td>
<td>Normal-10</td>
<td>2.35±1.11</td>
<td>60.5±13.35</td>
<td>138.64±63.47</td>
<td>58.7±9.61</td>
<td>73.2±7.84</td>
</tr>
<tr>
<td></td>
<td>Infertile-24</td>
<td>1.77±0.70</td>
<td>16.14±15.55</td>
<td>27.00±25.56</td>
<td>17.33±15.00</td>
<td>46.58±22.12</td>
</tr>
</tbody>
</table>

**Table 2.** BMI and frequency of infertility condition among all male subjects.

<table>
<thead>
<tr>
<th>Conditions</th>
<th>BMI</th>
<th>&gt;20 (n=36)</th>
<th>21-25 (n=180)</th>
<th>26-30 (n=139)</th>
<th>&gt;30 (n=49)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (n=125)</td>
<td>14</td>
<td>54</td>
<td>47</td>
<td>10</td>
<td></td>
</tr>
<tr>
<td>A spermia (n=14)</td>
<td>2</td>
<td>6</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Azoospermia (n=101)</td>
<td>12</td>
<td>51</td>
<td>26</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Oligospermia (n=20)</td>
<td>-</td>
<td>8</td>
<td>11</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Asthenospermia (n=31)</td>
<td>1</td>
<td>13</td>
<td>11</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>OA (n=43)</td>
<td>3</td>
<td>17</td>
<td>18</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>OATN (n=48)</td>
<td>2</td>
<td>22</td>
<td>18</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>OAN (n=17)</td>
<td>2</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>OAT (n=5)</td>
<td>-</td>
<td>5</td>
<td>-</td>
<td>-</td>
<td></td>
</tr>
</tbody>
</table>

OA: Oligoasthenospermia OAT: Oligoasthenoteratospermia, OATN: Oligoasthenoteratonecrospermia, OAN: Oligoasthenonecrospermia

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