Kisspeptin levels in infertile male subjects with abnormal sperm parameters

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Madam, Kisspeptin is a neuropeptide precursor which is encoded by kiss1 gene. It comprises of 145 amino acids which is proteolyzed to various lengths to form kisspeptin-10, kisspeptin-13, kisspeptin-14 and kisspeptin-54. Kisspeptin-54 is the major fragment. These fragments share an RFamide at the carboxy terminal. Kisspeptin is expressed in the hypothalamus, arcuate nucleus, gonads, placenta, liver and pancreas. It binds to receptors KISS1R/1kiss1r with equal efficacy. Kisspeptin receptor is G-coupled protein receptor. Kisspeptin plays a prominent role in reproduction. It regulates hypothalamo-pituitary-gonadal axis and causes gonadotrophin release. It causes LH release and may be helpful in causing ovulation in cases of in-vitro fertilization cycles. The reproductive endocrinologists have determined the role of Kisspeptin in oocyte maturation and improvement in endometrial thickness in infertile females reported for Intracytoplasmic Sperm Injection (ICSI).

Increased prevalence of male infertility with the associated social and psychological problems has called the researchers to look for causes as well as elucidations of the infertility catastrophe. One of the discovered causes is decrease in serum levels of Kisspeptin in infertile males as compared to fertile ones. Talking about infertility and its characterization by normal sperm parameters; as per WHO criteria; Normozoospermic group has normal number, motility, and morphology, yet infertile due to unknown reasons. Teratozoospermia corresponds to all standard parameters except that morphology of sperms was less than the reference range. Oligozoospermic had low sperm counts which had further two subdivisions; Oligozoospermia coupled with abnormal motility, group was referred to as Oligoasthenozoospermia. Oligozoospermia with less than normal morphological features, was grouped as oligoteratozoospermia (OAT) and when all sperm characteristics were abnormal, the group was oligoasthenoteratozoospermia (SOAT). The objective of the study was detection of Kisspeptin in different subgroups of infertile population.

We therefore estimated the levels of Kisspeptin and all the hormones responsible for male fertility in an infertile male

Table: Comparison of Kisspeptin and Reproductive Hormones in infertile sub groups with reference to different Sperm parameters.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Normozoospermia (n=15)</th>
<th>Teratozoospermia (n=21)</th>
<th>Azoospermia (n=5)</th>
<th>Oligo Asstenoteratozoospermia (n=3)</th>
<th>Oligo Asthenozoospermia (n=5)</th>
<th>Oligoteratozoospermia (n=6)</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (Years)</td>
<td>32.2 ± 3.75</td>
<td>35.14 ± 7.56</td>
<td>29.13</td>
<td>36.33 ± 4.51</td>
<td>38.6 ± 9.3</td>
<td>38.17 ± 3.31</td>
<td>0.06</td>
</tr>
<tr>
<td>Total Count Millions/ml</td>
<td>97.93 ± 22.92</td>
<td>41.52 ± 16.48</td>
<td>29.73</td>
<td>33.33 ± 6.51</td>
<td>22.2 ± 5.89</td>
<td>25.83 ± 7.08</td>
<td>0.00</td>
</tr>
<tr>
<td>Motility % age</td>
<td>73.67 ± 7.92</td>
<td>26.84 ± 6.75</td>
<td>0 ± 0</td>
<td>33 ± 3.61</td>
<td>22.2 ± 5.89</td>
<td>25.83 ± 7.08</td>
<td>0.00</td>
</tr>
<tr>
<td>Normal morphology % age</td>
<td>0.11 ± 0.02</td>
<td>0.04 ± 0.02</td>
<td>0 ± 0</td>
<td>0.02 ± 0.01</td>
<td>0.05 ± 0.03</td>
<td>0.03 ± 0.01</td>
<td>0.00</td>
</tr>
<tr>
<td>BMI kg/m²</td>
<td>26.02 ± 3.74</td>
<td>26.84 ± 2.72</td>
<td>29.88 ± 6.33</td>
<td>27.46 ± 1.2</td>
<td>27.82 ± 4.32</td>
<td>27.01 ± 0.88</td>
<td>0.28</td>
</tr>
<tr>
<td>Kisspeptin ng/ml</td>
<td>8 ± 2.51</td>
<td>6.04 ± 1.29</td>
<td>6.62 ± 1.2</td>
<td>5.63 ± 1.74</td>
<td>5.09 ± 1.27</td>
<td>4.81 ± 0.45</td>
<td>0.00</td>
</tr>
<tr>
<td>Follicle Stimulating Hormone (IU/mL)</td>
<td>3.87 ± 2.07</td>
<td>2.45 ± 0.91</td>
<td>3.66 ± 0.8</td>
<td>1.86 ± 1.66</td>
<td>2.52 ± 0.23</td>
<td>2.12 ± 0.79</td>
<td>0.01</td>
</tr>
<tr>
<td>LH(IU/mL)</td>
<td>3.4 ± 3.04</td>
<td>5.73 ± 1.36</td>
<td>6.66 ± 0.32</td>
<td>4.97 ± 2.67</td>
<td>6.58 ± 0.26</td>
<td>6.33 ± 1.14</td>
<td>0.00</td>
</tr>
<tr>
<td>Testosterone ng/dl</td>
<td>2.91 ± 2.32</td>
<td>3.1 ± 1</td>
<td>1.82 ± 0.43</td>
<td>1.69 ± 0.51</td>
<td>2.1 ± 0.12</td>
<td>2.9 ± 0.74</td>
<td>0.29</td>
</tr>
<tr>
<td>SHBG</td>
<td>14.64 ± 3.06</td>
<td>10.77 ± 1.51</td>
<td>11.69 ± 3.25</td>
<td>11.84 ± 1.14</td>
<td>8.38 ± 3.48</td>
<td>13.92 ± 1.71</td>
<td>0.00</td>
</tr>
</tbody>
</table>

Values are Mean ± SD, Results compared by Analysis of Variance
Reference Values: Normal Sperm Count (106 per ml) 3933±46
Motility % age, 40 (38-42)
Normal Morphology (4%).

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population with recognized sperm parameters. Since these infertile subjects have altered sperm parameters; count, motility and morphology hence comparison of levels in these groups can give an information about the impact of Kisspeptin on different sperm parameters. The highest level of Kisspeptin in normozoospermic males as compared to oligo teratozoospermic which had low sperm count and normal morphology (Table) in this pilot study directs us to further investigate and compare the impact of Kisspeptin on male reproductive axis by its impact on sperm count, motility and morphology in fertile and infertile males on a bigger sample of population.

We are hopeful that sequential research in this topic with promising results will enable us to look forward for finding more about the impact of Kisspeptin on male infertility as well as new avenues for the treatment of male infertility in specific subgroups of male infertile population.

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**References**