ABSTRACT

Hyperprolactinemia is characterized with a galactorrhea and menstrual dysfunction. Previous studies on this subject have indicated that serum prolactin level is increased by hypothalamic thyrotrophin-releasing hormone (TRH). Serum thyrotrophin level (TSH) is usually measured concerning to diagnosis and treatment, since hypothyroidism can induce galactorrhea. Moreover, it has been put forward that the effects on ovulal follicular maturation and corpus luteum function were observed in hyperprolactinemic women.

In this study, serum TSH, follicle-stimulating hormone (FSH) and luteinizing hormone (LH) levels were determined by immunoradiometric assay (IRMA) in hyperprolactinemic women with galactorrhea and menstrual dysfunction. TSH, FSH and LH levels of hyperprolactinemic subjects were compared to healthy control group. Our study groups were not receiving any drug such as oral contraceptives, bromocriptin and thyroid.

The results showed that FSH and LH were significantly (p<0.05) lower than the controls, while the difference for TSH level was not significant (p>0.05). In conclusion, it can be postulated that additional FSH determination together with TSH, FSH and LH may be considered in galactorrhea and amenorrhea cases.

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INTRODUCTION

Hyperprolactinemia is characterized by galactorrhea and menstrual dysfunction. Disorders of the neuroendocrine system regulating ovarian function generally result in anovulation and amenorrhea (1,2). Sustained hyperprolactinemia has been reported to interfere with ovarian follicular maturation and to impair corpus luteum function (2,3). Furthermore, it has been put forward that elevated serum PRL (prolactin) reduces the frequency of LH (GnRH) secretion by increasing hypothalamic opioid activity and suggest that the anovulation in hyperprolactinemia is consequently upon persistent slow frequency LH secretion (4-6).

In addition, that thyroid hormone may have an effect on the secretion of PRL has been proposed for a long time (7). Direct evidence for an inhibitory effect of thyroid hormones on PRL synthesis and release has been obtained in in vitro studies (8-10). It well established that TRH (Thyrotropin-releasing hormone) exerts a stimulatory effect on the secretion of both TSH and PRL (7,11). Clinically, hyperprolactinemia is usually presented in hypothyroid women (8). Increased pituitary responsiveness to TRH may be underlying cause of the increase in PRL secretion and it can be corrected by thyroid hormone replacement (11).

In this study, we sought to determine whether there are significant changes in serum TSH, FSH and LH levels in hyperprolactinemic women when compared with those from normoprolactinemic control subjects.

MATERIALS AND METHODS

Twenty-two hyperprolactinemic women with the complaints of galactorrhea and menstrual dysfunction were included in this study. Twenty healthy women, aged 18-39 years, with normal regular menstrual cycles served as controls. All cases and controls were not given any hormone or medicine during the study. Blood samples were obtained daily between 9-11 a.m. before breakfast from hyperprolactinemic women and controls. Immediately after sampling, serum was separated after centrifugation and stored at -20°C until assay.

Radioimmunoassays (RIA), were performed using commercially available kits for serum PRL, FSH, LH, T3, T4 and TSH levels (Diagnostic Products Corporation USA). The significance of differences was tested by student’s t test for the hormonal levels, with p < 0.05 regarded as statistically significant.

RESULTS

Serum PRL, FSH, LH, TSH and thyroid hormone levels in the patients and controls are shown in Table 1.

Table 1:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Hyperprolactinemic Women</th>
<th>N=22</th>
<th>X±SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prolactin (ng/ml)</td>
<td>89.2±63.5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>FSH (iu/ml)</td>
<td>5.5±4.8</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LH (iu/ml)</td>
<td>2.1±1.7</td>
<td></td>
<td></td>
</tr>
<tr>
<td>TSH (iu/ml)</td>
<td>1.52±1.01</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T3 (ng/ml)</td>
<td>147.3±122.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>T4 (ug/dl)</td>
<td>9.8±2.9</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Not significant (p>0.05, Student’s
galactorrhea and menstrual
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orrhea (1,2). Sustained
rhea with ovarian follicular
Furthermore, it has been
ed frequency of LH
activity and suggest that the
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an effect on the secretion of
idence for an inhibitory effect
has been obtained in in vitro
yropin-releasing hormone) SH
and PRL (7,11). Clinically,
roid women (8). Increased
cause of the increase in PRL
replacement (11).
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cycles served as controls. All
medicine during the study.
ly after sampling, serum was
using commercially available
levels (Diagnostic Products
was tested by student's t test
istically significant.

<table>
<thead>
<tr>
<th>PARAMETERS</th>
<th>Hyperprolactinemic Women</th>
<th>Control Group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n=22 X:SD</td>
<td>n=20 X:SD</td>
</tr>
<tr>
<td>Prolactin (ng/ml)</td>
<td>89.2±63.5</td>
<td>12.6±5.2</td>
</tr>
<tr>
<td>FSH (μU/ml)</td>
<td>5.5±4.8</td>
<td>10.7±5</td>
</tr>
<tr>
<td>LH (μU/ml)</td>
<td>2.1±1.7</td>
<td>5.5±4.7</td>
</tr>
<tr>
<td>TSH (μU/ml)</td>
<td>1.52±1.01</td>
<td>1.70±1.02</td>
</tr>
<tr>
<td>T3 (ng/ml)</td>
<td>147.3±32.2</td>
<td>143.0±25.7</td>
</tr>
<tr>
<td>T4 (μg/dl)</td>
<td>9.8±2.9</td>
<td>9.6±1.0</td>
</tr>
</tbody>
</table>

* Not significant (p>0.05, Student's t test)
Serum prolactin levels in the patient group were above 50 ng/ml (except two patients. ~35 ng/ml). There was highly significant difference (p<0.0001) between the patient and control group in terms of PRL.

The levels of FSH (p<0.05) and LH (p<0.05) in hyperprolactcinemic cases were found to be lower than those of controls, whereas there were no statistically significant differences in terms of TSH and thyroid hormones (p<0.05).

DISCUSSION

In the present study, 22 galactorrhoeic women with menstrual dysfunction had elevated periovulatory serum PRL levels. Previous studies suggested that luteal phase deficiency could be attributed to impaired folliculogenesee due to suppression of FSH levels during the follicular phase, attenuated LH surge, or elevated PRL secretion (4,5,14).

Recently, attempts have been made to characterize the neuroendocrine status of women affected by amenorrhoea, and some aberrations in hypothalamic-pituitary axis function have been described (15,16).

We have observed in our study low levels of FSH and LH in hyperprolactcinemic women with amenorrhoea and galactorrhoea as compared them to control group. Cok et al. have also pointed out reduced level of FSH and LH as well as anovulation in hyperprolactcinemic women (4).

Some clinical observations have displayed significantly often hyperprolactcinemia which were observed in primary hyperthyroid cases that are together with increased TSH levels (7). However, in our study we have not observed significantly different values in both serum TSH and thyroid hormones (T3, T4) in hyperprolactcinemic women. The other study has put forward that effect of thyroid hormone to prolactin secretion was observed only in case of dysfunction of thyroid (9). Hyperprolactcinemia primarily depends on age of patient and progress of illness in hypothroyd cases (3,8).

As thyroid hormone is given in hypothyroid cases represents thyrotropic hyperplasia, all changes such as galactorrhoea, amenorrhoea, gynecomastia and including hyperprolactcinemia return to normal. Conversely, thyropic tumor cases do not show any response to treatment (8-11).

In summary, the results showed that serum FSH and LH levels of hyperprolactcinemic women were significantly lower than the controls, while the difference for TSH level was not significant. In conclusion, it can be postulated that additional other pituitary hormones (such as TSH, FSH and LH) determinations together with PRL may be considered in galactorrhoea and amenorrhoeic cases.

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