Effects of neonatal Thyroid Stimulating hormone and free T4 levels on Apgar score, Maturity and Modes of delivery

Authors
Tabassum Yasmin¹, Shafat Imam Siddiqui², K.R. Prasad³
Naved Ahmad⁴, Akash Gupta⁵*

1MD, Assistant Professor, Department of Biochemistry, Rama Medical College and Hospital, Pilakhua, Hapur, U.P.
2MD, Assistant Prof, Department of Medicine, Katihar Medical College, Katihar, Bihar
3MD, Professor, Department of Biochemistry, Katihar Medical College, Katihar, Bihar
4PhD scholar, Department of Biochemistry, Subharti Medical College, Meerut, U.P.
5*MD, Assistant Professor, Department of Biochemistry, Rama Medical College and Hospital, Pilakhua, Hapur, U.P.
*Corresponding Author

Dr Akash Gupta
Department of Biochemistry Rama Medical College and Hospital, Pilakhua, Hapur, U.P
Email - akash_inspace@yahoo.com

Abstract
Thyroid disorders are four to five folds more common in women particularly during child bearing period. Cord blood is a source for the determination of thyroid related hormones in neonates. To find the effect of TSH and fT4 levels on apgar score of neonates, on term and preterm delivery and on mode of delivery. Total 80 pregnant and 80 non pregnant females were selected and their serum TSH and fT4 levels and cord blood of new born were estimated by ELFA technique on Minividas automated analyser. In our study we have found that there was no significant correlation of serum TSH and fT4 between the cases and controls of both the age groups, between asphyxiated and non asphyxiated neonates and the neonates born by vaginal delivery or caesarean section. Serum TSH was found to be significantly higher in the preterm neonates as compared to term neonates. Our study suggests that TSH has influence on maturity of neonates born to pregnant mothers. But TSH and fT4 does not have any influence on Apgar score and mode of delivery

Keywords – TSH, fT4, Cord Blood, Apgar score, Mode of delivery
Introduction

Around 42 million Indians are suffering from thyroid disorders and about 200 million people are at risk of iodine deficiency disorders. [1,2] Thyroid disorders are more frequent in women of child bearing age. [3] Thyroid hormones deficiency during gestation is associated with permanent brain damage. [4-6] Cord blood is a source for the determination thyroid related hormones in neonates. [7] The postnatal surge in TSH levels, common to all newborn is considered to be mediated through alpha adrenergic stimulation following the cold stress. [8] This alpha adrenergic stimulation in turn might be responsible for the observed increase in cord blood TSH in subjects who had low Apgar score, required active resuscitation after birth were born through vaginal delivery or non-elective lower section caesarean section and to primiparous mother. [9] Neonatal exposure to cold external environment at time of the parturition may evoke a marked and transient elevation of TSH. [10] Asphyxia cause both anatomical as well as physiological damage of different organs. Different causes of fetal hypoxia, before and during birth results in gestational age specific neuropathy. A better understanding of the maternal fetal inter relationships related to the ongoing thyroid process must remain our constant quest.

Materials and methods

The present hospital based study was undertaken in the department of Biochemistry in collaboration of the department of Obstetrics and Gynaecology and paediatrics, Katihar Medical College and Hospital, Bihar. A total of 80 pregnant women (cases) and 80 non pregnant women (controls) accompanying the pregnant women were selected. Both, cases and controls were not on any medication at the time of investigations. The cases and controls were informed about the risk and benefit of the study. Written consent was taken from all the subjects. The study was approved by institutional ethical committee. The new born of the pregnant mother were divided into two groups of Apgar score < 4 (asphyxiated) and ≥ 4 (non-asphyxiated). The study was extended to find out the effect of mode of delivery (vaginal or cesarian) and maturity (term or preterm) on pituitary thyroid axis of new born of above mothers. Venous blood of pregnant mother and cord blood of new born of these mothers was collected and allowed to clot to obtain the serum. Estimation of serum fT4 and TSH was done by enzyme linked fluorescent technique on Miniidas instrument from Biomerieux, France. Reference range of TSH is - 0.34 to 4.25 µIU/ml and of fT4 is - 0.7 to 1.24 ng/dl. [11]

Statistics

Data was analysed using the statistical package programme (SPSS) ver15. The data for biochemical analysis were expressed as mean ± SD and p value of < 0.05 was considered as significant.

Results

Our study was based on 160 subjects, out of which 80 were pregnant ladies (cases) and 80 were non pregnant ladies (controls). Mean ± SD
values of TSH and fT4 of cases and controls having age up to 30 years and > 30 years are shown in Table 1 and 2 respectively. In our study we have found that there was no significant correlation of serum TSH and fT4 between the cases and controls of both the age groups.

Also, there was no significant correlation of serum TSH and fT4 between asphyxiated and non-asphyxiated neonates as shown in Table 3.

A significant correlation was found in serum TSH between preterm neonates and term neonates and the value of serum TSH was found to be significantly higher in the preterm neonates as compared to term neonates. But there was no significant correlation of serum fT4 between preterm and term neonates. (Table 4)

We have also compared serum TSH and fT4 between neonates delivered by vaginal route and caesarean section and no significant correlation was found. (Table 5)

Table 1. Comparison of TSH and fT4 values between the pregnant mothers and controls in the age groups up to 30 years.

<table>
<thead>
<tr>
<th></th>
<th>TSH (µIU/ml)</th>
<th>fT4 (ng/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case</td>
<td>2.84 ± 2.13</td>
<td>1.12 ± 0.51</td>
</tr>
<tr>
<td>Control</td>
<td>2.91 ± 1.17</td>
<td>1.08 ± 0.48</td>
</tr>
<tr>
<td>P value</td>
<td>0.68</td>
<td>0.41</td>
</tr>
</tbody>
</table>

Table 2. Comparison of TSH and fT4 values between the pregnant mothers and controls in the age group more than 30 years.

<table>
<thead>
<tr>
<th></th>
<th>TSH (µIU/ml)</th>
<th>fT4 (ng/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case</td>
<td>2.62 ± 1.31</td>
<td>1.19 ± 0.42</td>
</tr>
<tr>
<td>Control</td>
<td>3.10 ± 1.40</td>
<td>1.10 ± 0.50</td>
</tr>
<tr>
<td>P value</td>
<td>0.11</td>
<td>0.36</td>
</tr>
</tbody>
</table>

Table 3. Comparison of TSH and fT4 values between the asphyxiated and non-asphyxiated neonates.

<table>
<thead>
<tr>
<th></th>
<th>TSH (µIU/ml)</th>
<th>fT4 (ng/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asphyxiated</td>
<td>7.36 ± 6.50</td>
<td>2.61 ± 0.93</td>
</tr>
<tr>
<td>Non asphyxiated</td>
<td>19.6 ± 11.50</td>
<td>3.11 ± 1.23</td>
</tr>
<tr>
<td>P value</td>
<td>0.95</td>
<td>0.03</td>
</tr>
</tbody>
</table>
Table 4. Comparison of TSH and fT4 values between the preterm and term neonates.

<table>
<thead>
<tr>
<th></th>
<th>TSH (µIU/ml)</th>
<th>fT4 (ng/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preterm neonates</td>
<td>18.05 ± 9.70</td>
<td>2.70 ± 0.87</td>
</tr>
<tr>
<td>Term neonates</td>
<td>7.84 ± 7.90</td>
<td>3.08 ± 1.26</td>
</tr>
<tr>
<td>P value</td>
<td>0.00</td>
<td>0.06</td>
</tr>
</tbody>
</table>

Table 5. Comparison of TSH and fT4 values between the babies delivered by vaginal route and caesarean section.

<table>
<thead>
<tr>
<th></th>
<th>TSH (µIU/ml)</th>
<th>fT4 (ng/dl)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vaginal delivery</td>
<td>11.39 ± 12.95</td>
<td>3.38 ± 1.63</td>
</tr>
<tr>
<td>Caesarean section</td>
<td>10.11 ± 17.39</td>
<td>2.98 ± 0.93</td>
</tr>
<tr>
<td>P value</td>
<td>0.68</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Discussion

Various authors have correlated an increase in TSH values with factors like birth asphyxia and difficult deliveries,\textsuperscript{[12]} perinatal stress events,\textsuperscript{[13]} instrumental delivery\textsuperscript{[14]} and negatively with cesarian sections as mode of delivery.\textsuperscript{[15]}

Rashmi et al\textsuperscript{[12]} reported that infants with birth asphyxia (Apgar score < 4 at 5 minutes) had significantly higher cord blood TSH levels (mean 31 µIU/ml, n=18) as compared to those without (mean 10.4 µIU/ml) (p < 0.01). The highest TSH levels were noted in neonates delivered by forceps extraction (mean 29.4 µIU/ml, n=17) and lowest levels in infants born by elective caesarean section (mean 8.7 µIU/ml, n=149).

Suloviv V et al\textsuperscript{[16]} reported that the serum T3 concentration in cord blood was significantly lower than that in the serum of the mother. However contrary to T3, rT3 in cord serum was significantly higher than the maternal serum. Also, the level of TSH in cord blood was considerably higher than in maternal serum.

Avruskin et al\textsuperscript{[17]} in their study found that total T3, T4 and TBG levels were all significantly higher than in normal non pregnant women in all trimesters (p <0.001). TSH levels remained unchanged in all three trimesters; values were 2.2, 2.0 and 2.0 µIU/ml respectively. Free T4 levels were at lower limits (2.4, 2.5 and 2.2 ng/dl) and free T3 levels were below normal (0.29, 0.27 and 0.24 ng/dl). Maternal and cord fT3 and fT4 levels were not significantly different.

Pereira et al\textsuperscript{[18]} reported in their study that gestational age, birth weight, sex, size for gestational age, mode of delivery and skin colour (white and non-white) were similar for both groups ( with Apgar score < 3 and >8). No differences were found in mean levels of cord...
blood TSH, T4, T3 and fT4 between the groups. In the samples collected 18 to 24 hrs after birth, mean levels of TSH, T4, T3 and fT4 were significantly lower in the asphyxiated group than in the control group. Mean concentrations of arterial TSH, T4 and T3 between 18 and 24 hrs of life were lower than concentrations found in the cord blood analysis in asphyxiated new born, but not in controls.

**Conclusion**

Pregnancy is a physiological condition that causes profound alteration of metabolic as well as endocrine environment. But there is a beautiful adaptation and so in most pregnant women, thyroid profile maintains its level within the non-pregnant reference range. Though the cord blood is an immediate source for the determination of thyroid related hormone in neonate, it is not a good specimen for knowing the thyroid status of the babies. Our study suggests that TSH has influence on maturity of neonates born to pregnant mothers. But TSH and fT4 do not have any influence on Apgar score and mode of delivery.

**References**

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