Short communication: Urine iodine concentration in neonates with congenital hypothyroidism

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Abstract

To examine the possible role of iodine in the aetiology of congenital hypothyroidism, urinary iodine concentration was compared in 23 neonates with congenital hypothyroidism detected by neonatal screening program in Riyadh region (mean cord TSH 403 ±123 mU/L), and their mothers. Twenty-five normal neonates (mean cord TSH 6.74 ± 4.03 mU/L) were randomly selected as control. The mean urine iodine concentration of the congenital hypothyroid group was 21.3 ± 11.2 μg/dl, which was significantly (P<0.05) higher than their mothers with mean of 16.3 ± 7.5 μg/dl, and the control group; mean 14.4 ± 4.6 μg/dl. We conclude that neonates with congenital hypothyroidism have normal iodine concentration, and that iodine does not seem to play a role in the aetiologial diagnosis of congenital hypothyroidism.

Keywords: congenital hypothyroidism, urine iodine, Saudi Arabia

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Introduction

The thyroid is unique among the endocrine glands for its dependence on an essential micronutrient, iodine, for normal hormone production [1]. The data presently available indicate that approximately 1.5 billion people are at risk of iodine deficiency disorders (IDD) [2]. Iodine deficiency, therefore, constitutes a major public health issue. The major threat is developmental cerebral disorders due to thyroid hormone deficiency in the fetal state (a combination of low transplacental transfer of hormone from the iodine deficient mother and insufficient hormone production from the iodine deficient fetal thyroid), and during the first years of life [3].

Urinary iodine estimation has been the traditional definitive method for assessment of iodine status in a population. Median urinary iodine levels in a population below 10 μg/dl indicate iodine deficiency [1-5].

In Saudi Arabia, where the incidence of congenital hypothyroidism is relatively high [6] urinary iodine excretion was not extensively studied. Stubbe et al [7] have indicated that the Saudi children in Riyadh region have normal urinary iodine excretion rate and this has been ascertained as well by Al Nuaim et al [8].

In this study, we are trying to assess the iodine status of neonates with congenital hypothyroidism and their mothers, and compare it with an age matched normal newborns in an attempt to examine the role of iodine in the aetiology of congenital hypothyroidism.

Materials and Methods
Urine (10 ml) was bag collected from 23 consecutive neonates who were diagnosed to have congenital hypothyroidism detected by neonatal screening program, Riyadh region (mean cord TSH 403 ± 123 mU/L). Values were confirmed by venous blood samples as recommended by the protocol. The mean recall TSH was 438 ± 148 mU/L. Thyroid Tc99m scan was performed (after urine collection) for aetiological classification. Urine samples (20 ml) were also collected from mothers of babies with congenital hypothyroidism in a plain tube. Twenty-five normal newborns (mean cord TSH 6.74 ± 4.03 mU/L) were randomly selected as control. Urine samples were stored at 4°C. Iodine concentration was calculated by the selective electrode method described by Pungor. Quality control of the assay was checked by using known iodine concentrations. The intra and interassay coefficient of variation (CV) were 11.7% and 11.2%, respectively for 30 μg/dl and 20 μg/dl, respectively.

Statistical analysis was done using Statpack Gold Statistical Analysis package. Data are presented as median and mean (SD). Student’s test was used to compare variable. P-values <0.05 was considered as significant.

Results

Urine iodine concentrations in the different groups are shown in Table 1. Although, the values for urine iodine concentration in the congenital hypothyroid group were within normal range; median 19.1 μg/dl and mean 21.3 ± 11.2 μg/dl, yet this is significantly (P<0.05) higher than that for their mothers and the control group. Table 2 shows the urine iodine concentration in the various congenital hypothyroid groups. All values are significantly (P<0.001) higher than the control group.

Table 1: Urine iodine concentration in neonates with congenital hypothyroidism (n=23), their mothers and control (n=25)

<table>
<thead>
<tr>
<th>Urine Iodine Concentrated(μg/dL)</th>
<th>Mean (SD)</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congenital hypothyroid neonates</td>
<td>21.3 (11.2)</td>
<td>19.1</td>
</tr>
<tr>
<td>Mothers of congenital hypothyroid neonates</td>
<td>16.3 (7.5)</td>
<td>15.4</td>
</tr>
<tr>
<td>Normal neonates (control)</td>
<td>14.4 (4.6)</td>
<td>13.9</td>
</tr>
</tbody>
</table>

Table 2: Urine iodine concentration in 23 neonates with congenital hypothyroidism according to aetiology

<table>
<thead>
<tr>
<th>Aetiological Classification (number)</th>
<th>Urine Iodine concentrated (μg/dL)</th>
<th>Mean (SD)</th>
<th>Median</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ectopic thyroid (13)</td>
<td>20.5 (10.3)</td>
<td>19.1</td>
<td></td>
</tr>
<tr>
<td>Dyshormonogenesis (6)</td>
<td>21.2 (8.5)</td>
<td>22.0</td>
<td></td>
</tr>
<tr>
<td>Aplastic thyroid (4)</td>
<td>27.0 (15.1)</td>
<td>21.3</td>
<td></td>
</tr>
</tbody>
</table>

Discussion

Although iodine is an essential element in thyroxine synthesis, it has very interesting contradicting effects in thyroid physiology. In physiological doses, up to 200 μg/day, it is necessary for hormone synthesis. In the contrary, supraphysiological exposure may exert antithyroid effects [1-3,10-15]. The risk of transient hypothyroidism in the neonates increases in the iodine deficient areas [2,11,12]. Furthermore, the severity of iodine deficiency in a given population might be reflected on the distribution of TSH values obtained in neonatal TSH screening programme for congenital hypothyroidism [10-13].
Congenital hypothyroidism, an important preventable cause of mental retardation is prevalent worldwide [6]. The majority of which are sporadic in nature, either aplasia, hypoplasia or ectopic with unknown aetiology so far. To our knowledge, the role of iodine in the aetiology of permanent congenital hypothyroidism has not been studied. The main objective of the present study was to assess the iodine status in neonates detected to have congenital hypothyroidism and their mothers in an attempt to delineate the interrelationship between iodine and permanent congenital hypothyroidism. Our results indicated that both neonates with congenital hypothyroidism and their mothers have sufficient iodine status and confirm previous local studies findings [7,8]. This indirectly suggests that iodine, per se, does not seem to play a role in the aetiology of congenital hypothyroidism. Of interest is the finding of higher mean values in neonates with thyroid aplasia as compared with those with thyroid ectopy and dyshormonogenesis. Although, the sample is small, it might reflect the absence of iodine utilization in that group, and indicates the need for further studies on large series of patients.

In conclusion, the present study indicates that our neonates with congenital hypothyroidism whose mothers have normal iodine status have normal iodine concentration. Iodine, per se, does not seem to play a role in the aetiological diagnosis of congenital hypothyroidism.

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References


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