Clinicopathological Observations on Calves with Enzootic Goiter
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It is a well known fact that enzootic goiter in farm animals has occurred in iodine deficient areas, and neonatal death frequently results if no suitable treatment is taken to correct the iodine deficiency [1, 9]. In Japan, there are few reports referred on goiter in farm animals [3, 6, 7, 8]. In this paper, we describe mainly the clinicopathological changes in calves affected with enzootic goiter which resulted in neonatal death or weakness.

The affected cases with goiter were eight Holstein calves born on three farms in Tokachi District of Hokkaido (Table 1). On farm N, 24.7% (28/113 calves) and 19.4% (21/114 calves) of newborn calves died of unknown cause between 1986 and 1987, respectively. Then, some of them with neonatal death showed enlargement of thyroids by postmortem examination and 4 calves (Nos. 1, 2, 3 and 4) of them were examined in this study. But, the enlarged thyroids in 4 calves were not apparently palpable. On the calf No. 5 at the age of ten days in farm O, the thyroid was enlarged to the size of a fist and recovered by iodine treatment. On farm S, 7.5% (3/40 calves) of feedlot calves at the age of twelve months (calf No. 6, 7, and 8) showed marked enlargement of thyroids almost to the size of a child’s head. The three calves with goiter commonly showed symptoms of tachypnea or dyspnea, and slaughtered.

The serum thyroxine (T₄) and triiodothyronine (T₃) levels of calves affected with goiter were determined by the RIA method and the results are shown in Table 2. Twenty-five healthy Holstein calves from 1 week to 12 months of age on a farm where goiter had not occurred were also examined as controls for serum T₄ and T₃ levels. Serum T₄ levels of goiter calves, except No. 2, indicates a decreasing tendency, 1.1 to 9.0 µg/100 ml, as compared with 13.3 ± 4.7 µg/100 ml of the control group. Serum T₃ showed remarkable high levels from 476 to 2,800 ng/100 ml in all cases as compared with 194 ± 47 ng/100 ml of the control group. Therefore, T₄/T₃ ratio was very low level of 0.5 to 19.3 as compared with 76.6 ± 19.4 of the control group. Especially, serum T₄ level of calf No. 6, which had the largest thyroid, was low of 1.4 µg/100 ml, and serum T₃ showed the highest level of 2,800 ng/100 ml among the examined calves.

In postmortem examinations, the thyroids of all the affected calves were bilaterally and diffusely enlarged. Figure 1 shows the marked enlargement of a thyroid (calf No. 6), and the construction of the trachea and esophagus had been caused by the enlarged thyroids.

Histologically, diffuse parenchymatous goiter due to hyperplasia of follicular epithelial cells was observed in all cases. The epithelial cells became cylindrically or columnnally shaped and protruded into the follicle through papillary proliferation. The thyroid of calf No. 1 is shown

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Table 1. Examined calves

<table>
<thead>
<tr>
<th>No.</th>
<th>Farm</th>
<th>Breed</th>
<th>Agea</th>
<th>Sex</th>
<th>Degree of thyroid enlargement</th>
<th>Clinical symptoms</th>
<th>Termination</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>N</td>
<td>Holstein</td>
<td>1d</td>
<td>Male</td>
<td>68 g</td>
<td>Tachypnea</td>
<td>Euthanasia</td>
</tr>
<tr>
<td>2</td>
<td>N</td>
<td>Holstein</td>
<td>1d</td>
<td>Male</td>
<td>30 g</td>
<td>Weakness</td>
<td>Neonatal death</td>
</tr>
<tr>
<td>3</td>
<td>N</td>
<td>Holstein</td>
<td>1d</td>
<td>Male</td>
<td>75 g</td>
<td>Weakness</td>
<td>Neonatal death</td>
</tr>
<tr>
<td>4</td>
<td>N</td>
<td>Holstein</td>
<td>1d</td>
<td>Female</td>
<td>50 g</td>
<td>Weakness</td>
<td>Neonatal death</td>
</tr>
<tr>
<td>5</td>
<td>O</td>
<td>Holstein</td>
<td>10d</td>
<td>Male</td>
<td>Fist size</td>
<td>Tachypnea</td>
<td>Recovery</td>
</tr>
<tr>
<td>6</td>
<td>S</td>
<td>Holstein</td>
<td>12 m</td>
<td>Male</td>
<td>Child’s head size</td>
<td>Dyspnea</td>
<td>Slaughtered</td>
</tr>
<tr>
<td>7</td>
<td>S</td>
<td>Holstein</td>
<td>12 m</td>
<td>Male</td>
<td>Child’s head size</td>
<td>Tachypnea</td>
<td>Slaughtered</td>
</tr>
<tr>
<td>8</td>
<td>S</td>
<td>Holstein</td>
<td>12 m</td>
<td>Male</td>
<td>Child’s head size</td>
<td>Tachypnea</td>
<td>Slaughtered</td>
</tr>
</tbody>
</table>

a) d: days, m: months.
Table 2. Serum thyroxine (T₄), triiodothyronine (T₃) levels and T₄/T₃ ratio in calves with goiter and control calves

<table>
<thead>
<tr>
<th>No.</th>
<th>T₄ (µg/100 ml)</th>
<th>T₃ (ng/100 ml)</th>
<th>T₄/T₃</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>4.5</td>
<td>680</td>
<td>6.6</td>
</tr>
<tr>
<td>2</td>
<td>17.9</td>
<td>925</td>
<td>19.3</td>
</tr>
<tr>
<td>3</td>
<td>9.0</td>
<td>540</td>
<td>16.7</td>
</tr>
<tr>
<td>4</td>
<td>1.1</td>
<td>476</td>
<td>2.3</td>
</tr>
<tr>
<td>5</td>
<td>2.2</td>
<td>724</td>
<td>3.0</td>
</tr>
<tr>
<td>6</td>
<td>1.4</td>
<td>2,800</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Control: 13.3±4.7 (µg/100 ml) 194±47 (ng/100 ml) 76.6±19.4 (T₄/T₃) (N=25) (4.9–23.5) (73–246) (51.6–118.6)

in Fig. 2. The follicles contained only little or no colloid and had irregular shapes because of the proliferated epithelial cells. The same findings were also observed in the cases Nos. 2, 3 and 4. Figure 3 (the thyroid of calf No. 6) showed more prominent hyperplasia of the epithelial cells than that in No. 1, and most of follicles were filled with the proliferated epithelial cells and the other follicles had irregular shapes. The cases of calf Nos. 5, 7 and 8 showed the same findings as calf No. 6.

From these findings, it was clarified that enzootic goiter of calves has occurred in Hok-
ENZOOTIC GOITER IN CALVES

Fig. 3. Excessive hyperplasia of follicular epithelium in markedly enlarged thyroid of No. 6. HE stain. ×100.

kaido. Moreover, it was confirmed that the goiter calves showed the reduction of T₄ and remarkable production of T₃, and suggested that the cause was disorders of the intake or utilization of iodine in the thyroid. Although the occurrence of goiter have been reported in iodine deficient areas [2, 4, 5], further investigation on the causes, such as the absolute lack of iodine or the intake of goitrogenic substances, should be carried out.

REFERENCES


要約

子牛の地方病性甲状腺腫に関する臨床ならびに臨床病理学的研究 (短報)：森永康裕，村野敏，更科孝夫，一条茂（帯広畜産大学畜産内科学教室）— 北海道のホルスタイン種子牛に地方病性甲状腺腫の発生が認められ，本病が子牛の生後直死や虚弱等の重要な原因となっていることが明らかになった。発病牛の甲状腺機能検査では，血中サイロキシシン (T₄) の低値と血中トリヨードサイロニン (T₃) の著明な上昇による T₄/T₃の低下を示し，病理学的所見では，甲状腺濁胞上皮細胞の過形成による機能性実質性甲状腺腫が共通して認められた。