CONGENITAL DIFFUSE HYPERPLASTIC GOITER ASSOCIATED WITH PERINATAL MORTALITY IN 11 CAPTIVE-BORN BOTTLENOSE DOLPHINS (TURSIOPS TRUNCATUS)


Abstract: Diffuse hyperplastic goiter was diagnosed by histopathology in 11 perinatal bottlenose dolphins (Tursiops truncatus) that died at four separate zoos and aquaria. Thyroid morphology of these animals was compared with the histologically normal thyroids of two stranded wild bottlenose dolphin calves, a neonate and a 2-mo-old calf. Histologic changes included reduced follicular luminal diameter, markedly reduced or absent luminal colloid, hypothyropathy of follicular epithelium, and follicular dysplasia. The etiology of the thyroid gland lesion was not identified. Cause of death was not determined for most of these animals, but they were presumed to have died from metabolic derangements associated with the thyroid lesion, drowning, or dystocia.

Key words: Bottlenose dolphin, Tursiops truncatus, goiter, thyroid gland.

INTRODUCTION

Congenital goiter can be acquired or inherited and is usually diffuse and hyperplastic rather than nodular or colloidial.1 Acquired diffuse hyperplastic goiter may occur with high or low maternal dietary iodine levels, the latter sometimes exacerbated by concurrent goitrogenic dietary substances.1,8,10 Acquired diffuse hyperplastic goiter is well documented in horses and domestic ruminants.1,7,8,10 Inherited dysmorphogenic goiter is the inability to synthesize and secrete adequate amounts of thyroid hormones beginning before or at birth, and it has been documented in human infants and as a heritable autosomal recessive trait in Corriedale, Dorset Horn, Merino, and Romney Marsh sheep, Afrikaner cattle, and Saanen dwarf goats.1 Thyroid lesions, including atrophy, adenoma, colloid goiter, and thyroiditis, have been described in captive and stranded dolphins,2,4,5,12 but perinatal diffuse hyperplastic goiter has not been described in dolphins or in other marine mammals. This report describes congenital diffuse hyperplastic goiter associated with perinatal mortality in 11 bottlenose dolphins (Tursiops truncatus) from four different facilities.

CASE REPORT

Signalment, calf and dam history, and geographic location are summarized in Table 1. Calves 1 and 2 were from the West Edmonton Mall Aquarium, Alberta, Canada. Calves 3 and 4 were from the Minnesota Zoo, calves 5–9 were from the Indianapolis Zoo, and calves 10 and 11 were from Dolphin Quest Hawaii, Hawaii, USA. Seven dolphins were males, and four were females. One dolphin was 6–8 wk premature, three were stillborn, six lived less than 1 day, and one lived 24 days. All but one dam were wild caught. Duration in captivity for the dams at the time of calf births averaged 11.5 yr, with a range of 6–23 yr. Dolphins 1 and 2, 3 and 4, and 5 and 6 had the same dam.

Dolphin facilities at the West Edmonton Mall and at the Minnesota and Indianapolis Zoos were closed water systems using synthesized salt water. The tank water was filtered through carbon and diatomaceous earth at the Edmonton facility and through sand and gravel filters at the Minnesota and Indianapolis Zoos. The tank water was chlorinated at Edmonton, and it was ozonated at the Minnesota and Indianapolis Zoos. The Dolphin Quest facility uses natural seawater in an open system.

All dolphins were necropsied within 24 hr of death. Representative sections of major tissues were preserved in 10% neutral buffered formalin. The tissues were processed routinely for histopathologic evaluation. Thyroid morphology of the affected
Table 1. Signalement and dams of neonatal bottlenose dolphin calves with congenital goiter.

<table>
<thead>
<tr>
<th>Calf</th>
<th>Location</th>
<th>Sex</th>
<th>Age</th>
<th>Weight (kg)</th>
<th>System</th>
<th>Dam, yr in captivity</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>West Edmonton</td>
<td>male</td>
<td>10 min</td>
<td>18.2</td>
<td>a</td>
<td>Maria, 11</td>
</tr>
<tr>
<td>2</td>
<td>West Edmonton</td>
<td>male</td>
<td>stillborn¹</td>
<td>13.2</td>
<td>a</td>
<td>Maria, 13</td>
</tr>
<tr>
<td>3</td>
<td>Minnesota Zoo</td>
<td>female</td>
<td>24 days</td>
<td>23.8</td>
<td>b</td>
<td>Mindy, 21</td>
</tr>
<tr>
<td>4</td>
<td>Indianapolis Zoo</td>
<td>female</td>
<td>&lt;1 day</td>
<td>11.3</td>
<td>b</td>
<td>Mindy, 23</td>
</tr>
<tr>
<td>5</td>
<td>Indianapolis Zoo</td>
<td>male</td>
<td>6 min</td>
<td>14.1</td>
<td>c</td>
<td>Ripley, 6</td>
</tr>
<tr>
<td>6</td>
<td>Indianapolis Zoo</td>
<td>female</td>
<td>stillborn</td>
<td>12.7</td>
<td>c</td>
<td>Ripley, 8</td>
</tr>
<tr>
<td>7</td>
<td>Indianapolis Zoo</td>
<td>male</td>
<td>15 min</td>
<td>16.8</td>
<td>c</td>
<td>Nova, 6</td>
</tr>
<tr>
<td>8</td>
<td>Indianapolis Zoo</td>
<td>male</td>
<td>stillborn</td>
<td>15.4</td>
<td>c</td>
<td>Shiloh, 6</td>
</tr>
<tr>
<td>9</td>
<td>Dolphin Quest</td>
<td>male</td>
<td>20 min</td>
<td>17.5</td>
<td>d</td>
<td>Phoenix, 11</td>
</tr>
<tr>
<td>10</td>
<td>Dolphin Quest</td>
<td>female</td>
<td>15 min</td>
<td>19.5</td>
<td>d</td>
<td>Poliathu, 10</td>
</tr>
<tr>
<td>11</td>
<td>Dolphin Quest</td>
<td>male</td>
<td>stillborn</td>
<td>14.5</td>
<td>d</td>
<td>Iwa, 24</td>
</tr>
</tbody>
</table>

¹ Closed system, salt water synthesized from road salt obtained from salt bed, filtered through carbon and diatomaceous earth mechanical filters, chlorinated; b: closed system, salt water synthesized by addition of salt, eight sand and gravel filters, ozonated; c: closed system, salt water synthesized by addition of salt, 12 sand and gravel filters, ozonated and chlorinated; d: natural seawater, open water system, no filters or treatment.

dolphins was compared with that of two wild-stranded bottlenose dolphins collected on the west coast of the Gulf of Mexico by one of us (DFC). One of these calves was estimated to be a few days old, and the other calf was estimated to be about 2 mo old.

Compared with the thyroid glands of the stranded dolphins, the thyroid glands of the affected calves were pale, had slight nodular irregularities, and had less bilateral symmetry. The thyroid glands of the stranded dolphins were similar to other standard mammalian thyroid histologic morphology and comprised discrete follicles lined by simple low cuboidal epithelium. The follicles contained abundant colloid (Fig. 1). Compared with the histologic morphology of the thyroid glands of the stranded dolphin calves and those of the previously described wild bottlenose dolphins⁵ and common (Pacific) dolphins, Delphinus delphis hemitænus,⁶ changes consistent with moderate to severe diffuse hyperplastic goiter were seen in all the captive-born dolphins. Goiter was the only consistent pathologic change seen in all the study dolphins. Thyroid gland changes (Figs. 2, 3) included reduced follicular luminal diameter or absence of lumina with associated reduction in luminal colloid, and hypertrrophy with vacuolation of follicular epithelium. Minimal to moderate dysplasia of the affected thyroid glands was characterized by mild crowding of follicular epithelium and loss of nuclear polarity, occasional fusion of follicles, and papillary infoldings of follicular epithelium. The interstitium between follicles often contained congested blood vessels. In some animals the morphologic changes were so prominent that the thyroid gland was not identified on gross examination. Additional histologic findings included lymphoid depletion consistent with stress in dolphin 2 and atrophy of fat consistent with suboptimal nutritional status in dolphin 8. Dolphin 3 had fibrinous degeneration of vessels walls, thrombosis, and fibrin deposition in the lung consistent with endotoxemia, and Vibrio alginolyticus was isolated from the lung.

**DISCUSSION**

Goiter in mammals is generally defined as hyperplastic or colloidal. Histologic changes associated with hyperplastic goiter include narrowing or obliteration of follicular lumina, reduction or absence of colloid, and hypertrophy or hyperplasia of follicular epithelium Colloid goiter is most often seen in adults and represents an involutionary phase of hyperplastic goiter. As hyperplastic follicular cells continue to produce colloid, endocytosis is decreased because of diminished pituitary thyroid-stimulating hormone, which occurs in response to return of blood thyroxine and triiodothyronine to normal. Subsequent histologic changes include marked distention of follicular lumina, flattening of follicular epithelium, and large amounts of colloid in the distended lumina. Based on the morphologic features of the thyroid gland lesions, the thyroid condition in these dolphins was classified as diffuse hyperplastic goiter.

Congenital diffuse hyperplastic goiter can be associated with nutritional or heritable disorders, or maternal exposure to goitrogenic compounds. High and low levels of dietary iodine have been docu-
mented to cause goiter in foals, ruminants, and various birds. The thyroid glands of the young may be exposed to higher levels of iodine than those of the dam, because iodine concentrates in the placenta and the mammary glands. It is possible that iodine levels in the food or water (or both) of these dolphins and their dams were suboptimal. Analysis of iodine levels in food and water is currently in process at the referring institutions.

The effect of goitrogens has been documented in mammals and birds. Goitrogenic compounds can disrupt thyrogenesis by impeding iodine metabolism in the dam, resulting in the development of goiter in the dam and the fetus. The water used in the dolphin exhibits was filtered, potentially removing microminerals. Ozone treatment of artificial seawater may alter the relative concentration of iodine species (iodide, iodate, and dissolved organic iodine) in a closed tank system. A number of potentially goitrogenic compounds may be present in the aquatic environment, including ammonia, nitrates, nitrites, and urea. The occurrence of hyperplastic goiter in the two calves maintained in an open seawater system suggests that factors other than goitrogens or water treatment may contribute to goiter development, such as suboptimal dietary iodine or impaired iodine metabolism in the dams.

Heritable diffuse hyperplastic goiter is documented in humans and in several breeds of cattle. All but one of the dams of the dolphins of our study were wild caught, and there was a wide demographic distribution of the animals, so it seems unlikely that these dolphins had a heritable disorder. The actual cause of death of these neonatal dolphins could not be determined histologically. Death was presumed to be due to metabolic derangements associated with thyroid dysfunction, drowning, or dystocia. Histologic changes associated with drowning or dystocia may be subtle or absent. We feel that drowning or dystocia may have contributed to the demise of some of the calves because no histologic lesions severe enough to be fatal (other than the thyroid gland lesions) were identified. Endotoxemia may have contributed to the deterio-
ration of dolphin 3 because histologic changes were consistent with sepsis. Atrophy of fat in stillborn dolphin 8 indicates suboptimal nutritional status in utero and may have been due to maternal or fetal illness or both.

We believe that the congenital goiter in these dolphin calves was an acquired disorder associated with derangements in the thyroid gland or iodine metabolism (or both). Maternal and fetal thyroid-derived hormone and iodine values may be altered by inappropriate levels of iodine in the diet or in the aquatic environment, or by exposure to one or more goitrogenic compounds in the environment. It is interesting to note that the dam of dolphins 1 and 2 subsequently died and had hyperplastic changes in the thyroid gland.

In addition to complete necropsy and histologic examination of all dead dolphins, ongoing studies at facilities where goiter has been diagnosed or suspected include evaluation of iodine and iodide levels in dolphin diets and exhibit water and comparison with known values for natural diets and seawater, respectively; evaluation of thyroid hormone values (total T4, free T4, total T3, free T3, reverse T3) and TSH levels for the affected neonatal and adult dolphins and comparison with known values for normal captive and wild dolphins; evaluation of thyroid gland to body weight ratios for the affected juvenile and adult dolphins and their comparison with values for healthy dolphins; and evaluation of tank water parameters for abnormal levels of potentially goitrogenic substances.

Acknowledgments: For slide preparation, we gratefully acknowledge the excellent technical support of Roy Brown, Histology Consulting Service, Oak Harbor, Washington. We thank Dr. B. E. Beck, Veterinary Pathology Laboratory, Edmonton, Alberta, Dr. Detlef Onderka, Animal Health Laboratories, Agriculture, Food and Rural Development,
Figure 3. Thyroid gland, neonatal dolphin with diffuse hyperplastic goiter. Follicles have fused to form a large pseudolumen containing blood and debris. Marked papillary infolding of the follicular epithelium lining the fused follicle (arrow) and focal crowding of the epithelium are present. H&E, bar = 95 μm.

Alberta, and Dr. David Gribble, Central Veterinary Laboratory, Vancouver, British Columbia, for providing slides and original diagnoses of dolphins 1 and 2. We also thank the Animal Disease Diagnostic Laboratory, Purdue University, West Lafayette, Indiana, for providing slides and original diagnoses of dolphins 5–7.

LITERATURE CITED


10. Silva, C. A., H. Merkt, P. N. Bergamo, S. S. Barros,


Received for publication 16 November 2001