



# Primary goitrous hypothyroidism in a young adult domestic longhair cat: diagnosis and treatment monitoring

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## Abstract

**Case summary** Primary goitrous hypothyroidism was diagnosed in a 12-month-old cat examined because of small stature, mental dullness, severe lethargy, generalized weakness and gait abnormalities. Radiographs of the long bones and spine revealed delayed epiphyseal ossification and epiphyseal dysgenesis. Diagnosis of primary hypothyroidism was confirmed by low serum concentrations of total and free thyroxine (T4) with high thyroid-stimulating hormone (TSH) concentrations. Thyroid scintigraphy revealed severe enlargement of both thyroid lobes, as evidenced by a seven-fold increase in calculated thyroid volume above the reference interval. In addition, this bilateral goiter had an extremely high radionuclide uptake, about 10-fold higher than the normal feline thyroid gland. Treatment with twice-daily levothyroxine (L-T4), administered on an empty stomach, resulted in increased alertness, playfulness, strength and improvement in gait, as well as an increase in body length and weight. L-T4 replacement also led to normalization of serum thyroid hormone and TSH concentrations, and complete resolution of goiter.

**Relevance and novel information** Spontaneous hypothyroidism is rarely reported in cats, with congenital hypothyroidism in kittens diagnosed most frequently. Despite the fact that this cat was a young adult, it likely had a form of congenital hypothyroidism caused by dyshormonogenesis (defect in thyroid hormone synthesis) that led to compensatory development of goiter. In hypothyroid cats, treatment with L-T4 is best given twice daily on an empty stomach to ensure adequate absorption. Normalization of serum TSH and shrinkage of goiter, as well as improvement in clinical signs, is the goal of treatment for cats with goitrous hypothyroidism.

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## Introduction

Naturally occurring, adult-onset primary hypothyroidism is an extremely rare clinical disorder in cats, with only three reported cases diagnosed in the prime to senior stage of life (aged 5, 5 and 12 years, respectively).<sup>1–3</sup> However, congenital hypothyroidism (cretinism) is also rare but is one of the most common causes of disproportionate dwarfism in kittens and is much better characterized than the adult-onset form.<sup>4,5</sup> Of the 60 or so cats with congenital hypothyroidism that have been reported,<sup>6–20</sup> only two were older than 12 months of age at time of diagnosis,<sup>16,17</sup> whereas another two cats were diagnosed at 7 and 8 months of age, respectively.<sup>12,19</sup> All of the remaining cases were diagnosed as kittens, generally at 2–4 months of age. Of these cats with congenital hypothyroidism, about half were reported to have palpable enlargement of the thyroid (goiter), which is likely a compensatory response associated with an intrathyroidal

defect in thyroid hormone biosynthesis (dyshormonogenesis).<sup>6,8,10,11,15,19</sup> Whether this feline goiter shrinks or resolves with thyroid hormone replacement therapy is not clear, but some have suggested that goiter may persist despite adequate treatment.<sup>15</sup>

The purpose of this case study was to describe the diagnostic testing, treatment and long-term outcome of a young adult domestic longhair cat first diagnosed with spontaneous goitrous hypothyroidism at the age of

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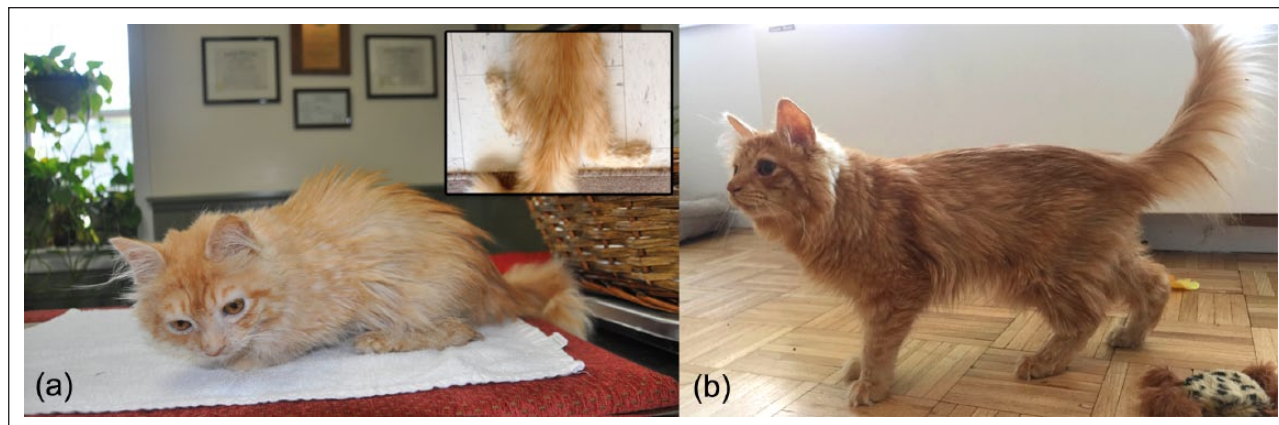
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**Figure 1** Young adult domestic longhair cat with goitrous hypothyroidism. (a) Cat at time of diagnosis (12 months of age) showing mental dullness, lethargy, small stature and unkempt hair coat; (inset) cat showing rear leg weakness, with splaying of the hindlegs into a frog-leg position on smooth surfaces. (b) Cat at 24 months of age after treatment with levothyroxine for 1 year

12 months (in the middle of the junior life stage<sup>21</sup>). For this cat, we used the clinical features, serum concentrations of thyroid hormone and thyroid-stimulating hormone (TSH), and results of both qualitative and quantitative thyroid scintigraphy to aid in both the diagnosis and long-term monitoring of thyroid hormone replacement therapy.

### Case description

A 12-month-old neutered male domestic longhair cat was examined for small stature, mental dullness, lethargy, inactivity, weakness and inability to jump or walk normally. The owner reported that the back legs tended to splay out on smooth flooring, although the cat could walk better on a rug or carpeted surface. The cat ate very slowly but the appetite was considered normal. No constipation or polyuria was reported. The cat had always been dull and inactive (never played, ran, jumped), but the weakness and gait abnormalities had progressively worsened over the past 10 months.

The owner had adopted the cat at 19 days of age as a stray kitten. At 5 weeks of age, scrotal and umbilical hernias were surgically repaired, and the kitten was also neutered at that time. Initially, the kitten was bottle-fed a commercial liquid milk replacer (KMR Liquid Milk Replacer; PetAg) and then transitioned over a period of a few weeks to a variety of flavors and brands of canned commercial cat foods (eg, Wellness Cat Food [WellPet LLC]; EVO [Natura Pet Products]; and Precise Naturals [Precise Pet Products]). The cat had continued to be fed this variety of commercial foods up to the time of initial evaluation.

On initial physical examination, the cat weighed 2.3 kg with a normal body condition score (3/9) and muscle condition score (3/3). The cat had an unkempt hair coat and was small in stature, with a body length of 34.5 cm from the tip of the nose to base of tail (Figure 1,

Table 1). The cat was severely lethargic and mentation was dull. The rectal temperature was 38.3°C, the heart rate 170 beats per minute and the abdomen palpated normally. Oral examination revealed normal adult dentition. Bilateral enlargement (goiter) affecting both thyroid lobes was detected on cervical palpation.

When encouraged, the cat could walk but did so in a crouched posture with a slightly arched back; the gait was slow, deliberate and short strided. The cat tired quickly and would lie down with the hindlegs splayed out in a frog-leg position (Figure 1a, inset). On neurologic examination, mental dullness, decreased menace response and generalized weakness were detected. No spinal, muscular or orthopedic pain could be elicited. Fundic examination revealed no abnormalities and the remainder of the neurological examination was unremarkable.

Radiography of the spine and rear legs revealed generalized delayed closure of the ossification centers of the long bones and vertebrae (Figure 2). This was considered inappropriate for a 12-month-old cat.<sup>22</sup> Blood was collected for a complete blood cell count, serum biochemical profile and total thyroxine (tT4) concentration. Hematologic evaluation demonstrated a mild normocytic, normochromic anemia, as evidenced by slightly low values for the red cell count, hemoglobin and hematocrit (Table 1). All results of the complete serum biochemistry analysis were within the respective reference intervals (RIs); however, the serum cholesterol concentration was at the high end of normal (5.46 mmol/l; Table 1). The serum tT4 concentration was low (6 nmol/l; RI 12–49 nmol/l). Based on the clinical signs and test results, hypothyroidism was suspected and the cat referred to the Animal Endocrine Clinic for further evaluation.

Physical examination again revealed a small, quiet cat with bilateral goiter. Serum for a complete thyroid panel was collected and tT4, free T4 by dialysis (fT4),

**Table 1** Serial measurements of body weight, serum thyroid hormone concentrations and quantitative thyroid scintigraphic parameters in a young adult domestic longhair cat with primary hypothyroidism

Parameter	Time since diagnosis (months)					Reference interval
	0	3	6	9	12	
Body weight (kg)	2.3	2.5	2.9	3.4	3.7	–
Body length (cm)	34.5	–	–	–	48.3	–
Red cell count ( $\times 10^{12}/l$ )	6.52	8.80	–	–	8.90	7.10–11.50
Hemoglobin (g/l)	99	120	–	–	128	103–162
Hematocrit (l/l)	0.29	0.42	–	–	0.42	0.30–0.52
Cholesterol (mmol/l)	5.46	3.42	–	–	5.35	1.95–5.70
Total T4 (nmol/l)	6	13	100	26	32	12–49
Free T4 (pmol/l)	8	12	90	–	31	10–51
Total T3 (nmol/l)	<0.5	0.6	1.8	–	0.6	<0.5–1.9
TSH (ng/ml)	4.63	0.30	<0.03	<0.03	<0.03	<0.03–0.30
Thyroid volume (g)	6.7	–	–	–	1.0	0.1–1.0
T/S ratio	7.9	–	–	–	0.9	0.5–1.5
TcTU (%)	14.70	–	–	–	0.25	0.05–0.80
L-thyroxine dose ( $\mu\text{g}/\text{day}$ )	0	100	150	100	100	–

T4 = serum thyroxine; TSH = serum thyroid-stimulating hormone; TcTU = percent thyroidal uptake of  $^{99\text{m}}\text{TcO}_4^-$ ; T/S = thyroid-to-salivary ratio; L-thyroxine = levothyroxine

triiodothyronine (T3) and TSH were determined by techniques previously validated for use in the cat.<sup>23</sup> These results confirmed the low tT4 concentration, but also revealed a low fT4 concentration and a markedly high TSH concentration (Table 1, month 0).

Thyroid scintigraphy was next performed by injecting 111 MBq of sodium  $^{99\text{m}}\text{Tc}$ -pertechnetate ( $^{99\text{m}}\text{TcO}_4^-$ ) intravenously and imaging an hour later, as previously described.<sup>24,25</sup> Quantification of thyroid activity was investigated by calculation of both the thyroid-to-salivary gland ratio (T/S) and the percent thyroidal uptake of the injected  $^{99\text{m}}\text{TcO}_4^-$  (TcTU).<sup>24–26</sup> The estimated thyroid volume was also calculated from the scintigraphic image, as previously described.<sup>25</sup> Analysis of the thyroid scintigram revealed both thyroid lobes to be greatly enlarged (Table 1), with the larger left thyroid lobe extending through the thoracic inlet well into the thoracic cavity (Figure 3a,b). The uptake of the radionuclide by the massive amount of goiter tissue was much higher than normal, as was confirmed by calculating high values for the T/S ratio and the percent TcTU (Table 1, day 0).

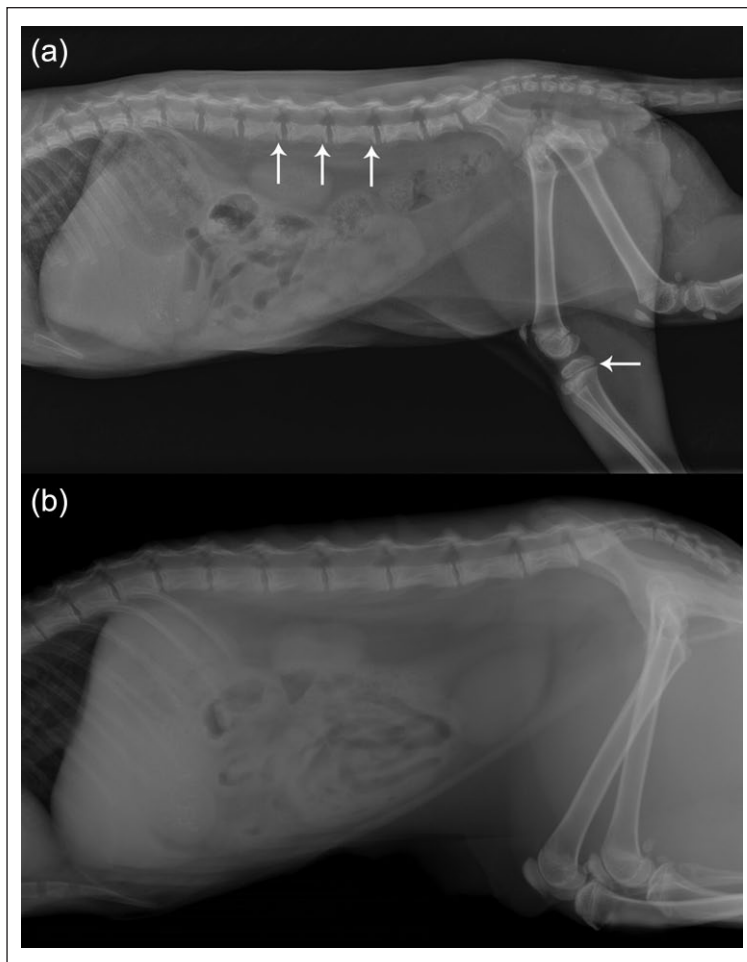
Overall, the clinical signs, laboratory results, radiological findings and scintigraphic findings were considered diagnostic for primary, congenital goitrous hypothyroidism. Treatment was initiated with an oral solution of levothyroxine (L-T4) (Leventa; Merck Animal Health) at a dose of 50  $\mu\text{g}$  PO q12h, administered on an empty stomach.

At the 3 month follow-up time, the cat was reported to be much less lethargic and more active. Weight gain and a subjective increase in body length were apparent

but the gait abnormality continued. The goiter remained palpable. Repeat hematological analysis revealed that the mild anemia had resolved, as evidenced by normalization of the total red cell count, hemoglobin concentration and hematocrit. Serum cholesterol remained within the RI, but the concentration had fallen to the middle of the RI (3.42 mmol/l). Follow-up endocrine testing revealed that tT4 and fT4 concentrations had risen to only the low end of the RI, whereas TSH had fallen but remained at the high end of the RI (Table 1; 3 months). Because of these endocrine results, the L-T4 dose was increased to 75  $\mu\text{g}$  q12h (Table 1).

At the 6 month follow-up, the cat was found to be more alert and active, with continued body growth and an increase in body weight (Table 1). Serum thyroid hormone monitoring now revealed that TSH concentration was suppressed but concentrations of both tT4 and fT4 concentrations were high. Therefore, the daily L-T4 dose was again reduced to 50  $\mu\text{g}$  q12h (Table 1).

At the 12 month follow-up treatment time (24 months of age), the cat was reported to continue to be more alert, interactive and affectionate. Although the cat could still not jump normally, the body posture and gait were much improved, and the cat was markedly stronger and did not tire easily. The cat had continued to gain weight and body length had increased 1.4-fold from the pretreatment measurement (Table 1). Serum thyroid hormone monitoring revealed normal serum tT4, fT4 and TSH concentrations. Repeat radiographs revealed complete closure of the physes of the vertebrae and long bones (Figure 2). Follow-up thyroid scintigraphy demonstrated



**Figure 2** Radiographs of a young adult domestic longhair cat with goitrous hypothyroidism. (a) Cat at time of diagnosis (12 months of age). Notice generalized delayed epiphyseal ossification (open physes) of the vertebra and long bones (arrows), as well as failure of fusion of the ischium, ilium and acetabulum, and poorly developed patellae. (b) Same cat at 24 months of age after treatment with levothyroxine for 1 year. Notice that the ossification centers (physes) of all vertebrae, long bones and pelvis are now closed and the patellae appear normal

a normal size and intensity of radionuclide uptake by both thyroid lobes, with complete resolution of the cat's intrathoracic goiter and normalization of thyroid uptake (Figure 3c, Table 1). At the time of this submission, the cat is 28 months old, has near-normal overall strength and gait, and is leading a relatively a normal life on an L-T4 dose of 50  $\mu\text{g}$  q12h (Table 1).

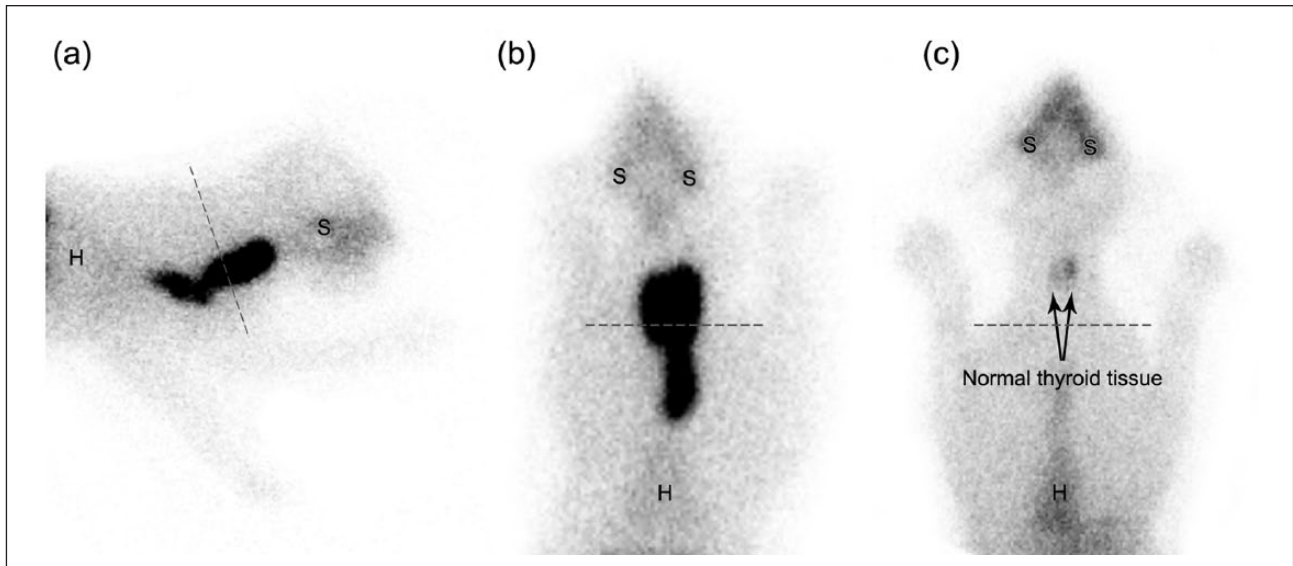
## Discussion

Although the cat of this report was not diagnosed until 12 months of age (during the junior life stage), it is likely that the underlying cause of the hypothyroidism was a congenital disorder rather than a spontaneous form of juvenile- or adult-onset hypothyroidism. Both the cat's small stature and delayed closure of the ossification centers of vertebra and long bones point to long-standing hypothyroidism, likely starting as a very young kitten.<sup>4,6-20</sup>

In addition, the fact that this cat had a palpable goiter indicates the presence of intact thyroid tissue (not characteristic for adult-onset hypothyroidism<sup>1-3</sup>), and the massive size of this goiter suggests a process that was ongoing for many months, most likely since birth. Acquired causes for goitrous hypothyroidism, such as iodine deficiency or environmental goitrogens, were considered highly unlikely based on the cat's life-long history and diet of a variety of commercial canned foods. We did not, however, test any of the commercial diets for iodine levels or goitrogens, so this still remains a remote possibility.

In cats, congenital hypothyroidism can result from a defect in thyroid gland development (ie, thyroid aplasia or hypoplasia) or a defect or a block in thyroid hormone production (dyshormonogenesis) by an anatomically intact thyroid gland. In this latter instance, thyroid dyshormonogenesis is associated with an inability to secrete adequate amounts of T4 and T3, which leads to the loss





**Figure 3** Thyroid scintigraphy in a cat with goitrous hypothyroidism, before and 1 year after treatment with levothyroxine (L-T4). Both scintigrams were obtained 1 h after intravenous administration of 110 MBq of sodium pertechnetate ( $^{99m}\text{TcO}_4^-$ ), as previously described.<sup>24,25</sup> (a) Thyroid scintigram (right lateral view) at time of diagnosis of hypothyroidism, before any thyroid hormone supplementation. The thyroid gland is greatly enlarged and extends through the thoracic inlet (indicated by dashed line) into the thoracic cavity. The uptake of the radionuclide by the hyperplastic goiter is much higher than normal, as seen by the increased intensity of uptake by the goiter compared with the salivary glands (indicated by 'S'). The radionuclide uptake in the normal thyroid closely approximates the uptake in the salivary glands, with an expected 'intensity' ratio of 1:1. The high radionuclide uptake in this cat was confirmed by calculating high values for the thyroid:salivary (T/S) ratio and the percent uptake of  $^{99m}\text{TcO}_4^-$  (TcTU; Table 1, day 0). (b) Thyroid scintigram (ventral view) at time of diagnosis. Both thyroid lobes are greatly enlarged with a homogeneous pattern of increased  $^{99m}\text{TcO}_4^-$  uptake. The larger left thyroid lobe extends through the thoracic inlet (dashed horizontal line) into the thoracic cavity. Again, the uptake of the radionuclide by the hyperactive goiter is much higher than normal, as seen by the increased intensity of uptake by the goiter compared with the salivary glands. (c) Ventral thyroid image of the same cat after supplementation with L-T4 for 12 months and normalization of serum concentrations of total thyroxine, free thyroxine (T4) and thyroid-stimulating hormone. The thyroid scintigram demonstrates dramatic shrinkage of the hyperplastic thyroid tissue with complete resolution of the large goiter. Both thyroid lobes are now within normal limits for size, shape and position in the cervical area. Radioactivity uptake in these thyroid lobes has decreased dramatically and now closely approximates activity in the salivary glands, with an expected T/S ratio of approximately 1:1 (Table 1). S = salivary gland; H = heart; dashed line = region of thoracic inlet

of normal negative feedback inhibition on pituitary thyrotropes with persistent secretion of excessive amounts of TSH. The unrelenting stimulation of intact thyroid follicular cells by the high circulating concentrations of TSH results in thyroid hyperplasia, enlargement of the intact thyroid and a clinically palpable goiter. Obviously, our cat had a form of thyroid dysmorphogenesis rather than aplasia or hypoplasia, based on the presence of goiter. The development of a palpable goiter in cats, dogs or humans with dysmorphogenesis takes time, however, and may be delayed in onset.<sup>27,28</sup> For example, in one family of affected kittens, goiter was not recognized within the first few weeks of life, but the enlarged thyroid could be readily palpated in all kittens by 5–6 months of age.<sup>10</sup> This again provides evidence that the massive amount of goiter tissue found in our cat likely indicates very long-standing hypothyroidism, starting most likely at time of birth.

A prominent clinical feature of hypothyroidism in this cat was the inability to jump or walk normally, which

appeared to be due to generally neuromuscular weakness. In human infants, congenital hypothyroidism can also cause a range of other neuromuscular sequelae, including abnormal muscle tone, severe muscle weakness, ataxia and motor incoordination.<sup>29,30</sup> Although the exact pathogenesis of these neuromuscular signs in our cat is not clear, marked improvement was evident with successful thyroid hormone replacement therapy.

In kittens and young juvenile cats, a presumptive diagnosis of congenital hypothyroidism can be made on the basis of clinical features (eg, dwarfism, mental dullness, open bone plates) and low serum tT4 concentrations.<sup>4,5</sup> However, the definitive diagnosis should never be based on a low tT4 alone, as many concurrent non-thyroid illnesses can also suppress the serum T4 concentration thus leading to a false-positive test result.<sup>31</sup> In addition, although the RIs for kittens may change as they move into their junior stage of life,<sup>32</sup> most laboratories have not established age-related RIs for the thyroid hormones, at least for kittens or juvenile cats. In the cat of this report,

we measured a complete serum thyroid profile, which included *t*T4, *f*T4, T3 and TSH concentrations. Our results, which showed low to low-normal thyroid hormone concentrations together with high levels of TSH, confirmed primary hypothyroidism.<sup>4,5,33</sup> We believe that finding high concentrations of TSH is the single most important endocrine test for diagnosis of feline hypothyroidism for two reasons: high TSH concentrations have been reported in all of the hypothyroid cats in which it was measured,<sup>2,3,9,15,18–20</sup> and falsely high values for TSH are not generally seen in cats with non-thyroidal illness.<sup>34</sup>

Thyroid scintigraphy was performed in our cat to better define the diagnosis and to differentiate thyroid aplasia or hypoplasia from dysshormonogenesis.<sup>35–37</sup> In cats with thyroid aplasia or hypoplasia, no thyroid tissue is visible on scintigraphy, and thyroid uptake of the radioisotope is low or undetectable.<sup>2,33</sup> In contrast, cats with dysshormonogenesis have an increased uptake of radioiodine into the thyroid gland,<sup>19,33</sup> as we documented in this cat by calculation of high values for the T/S ratio and TcTU (Table 1). Although obvious from the subjective assessment of the thyroid scintigram (Figure 3a,b), calculation of the cat's pretreatment thyroid volume also confirmed an approximately seven-fold increase above the RIs.

Cats with congenital hypothyroidism treated with L-T4 should have a relatively normal life expectancy, but the long-term prognosis may be dependent on etiology and age at initiation of treatment.<sup>27</sup> The findings in this cat indicate that even with delayed diagnosis, L-T4 treatment can improve or resolve most of the clinical signs, leading to a marked increase in overall body growth and an improvement in strength, activity and mentation. The final L-T4 dose needed to treat cats with hypothyroidism can vary tremendously, with some cats showing an apparent resistance to even relatively high doses (200–400 µg/day) and others (including our cat) responding well to lower doses (50–100 µg/day).<sup>3,13,14,16,19,20</sup> If resistance to L-T4 supplementation occurs, with persistently high serum TSH concentrations, it can be helpful to administer the dose on an empty stomach (as we did in our cat), as food can interfere with L-T4 absorption.<sup>38,39</sup> In addition, use of an oral solution of L-T4 (as we employed in this cat), rather than a tablet, may lead to an increased absorption of thyroid hormone.<sup>40,41</sup>

For monitoring of thyroid hormone supplementation in cats with congenital hypothyroidism, the L-T4 dose should be adjusted to normalize both serum *t*T4 and TSH concentrations. If the serum *t*T4 remains in the low-normal range and TSH remains high, the dose should be gradually increased and administered on an empty stomach, if possible. If the serum TSH falls but *t*T4 increases into the hyperthyroid range, the dose should be decreased, as overdosing can blunt the expected weight gain and growth.<sup>42</sup> With adequate treatment (and

normalization of high serum TSH concentrations), shrinkage of the goiter size is expected,<sup>43</sup> as we documented in this cat.

## Conclusions

In the cat of this report, diagnosis of primary hypothyroidism was confirmed by low serum concentrations of *t*T4 and *f*T4 with high TSH concentrations. Thyroid scintigraphy revealed severe enlargement of both thyroid lobes, as evidenced by a seven-fold increase in calculated thyroid volume above the RI. In addition, this bilateral goiter had an extremely high radionuclide uptake, about 10-fold higher than the normal feline thyroid gland. Despite the fact that this cat was a young adult, the author believes the cat had a form of congenital hypothyroidism caused by dysshormonogenesis (defect in thyroid hormone synthesis) that led to compensatory development of goiter. Treatment with twice-daily L-T4, administered on an empty stomach, resulted in increased alertness, playfulness, strength and improvement in gait, as well as an increase in body length and weight. Thyroid hormone replacement also led to closure of the open epiphyseal (growth) plates, normalization of high serum TSH concentrations and complete shrinkage of the goiter.

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