Congenital hypothyroidism in a cat

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An 18-month-old, 2 kg, intact female domestic shorthair was presented for a 2-day history of decreased appetite and lethargy. The owner had also noted that there was a decrease in the amount of urine and stool being passed. Further conversation revealed a history of chronic intermittent constipation since the age of 8 wk.

On examination (day 0), the cat was found to be very small in stature with a round body shape, short legs and neck, and a large, broad head (Figure 1). Deciduous teeth were still evident in the dentition and the coat was soft and fluffy. Abdominal palpation revealed an empty colon and a very small bladder. Rectal body temperature was 37.7°C and the pulse 180 beats/min. She was estimated to be 3% to 5% dehydrated on skin tent.

Blood was taken for a complete blood cell (CBC) count; biochemical profile, including electrolytes; and a total T4 and free T4 by equilibrium dialysis. An elevated blood urea nitrogen (BUN) and creatinine greater than 3 times the normal value, with a urine concentration of 1.010, supported a diagnosis of acute renal failure. Subsequent failure to produce urine following IV administration of 0.9% NaCl fluids suggested oliguric renal failure. Dopamine HCl (Inotropin; Dupont Pharma, Mississauga, Ontario) was added to the IV fluids at a rate of 2 μg/kg bodyweight (BW)/min, along with furosemide (Lasix 5% solution; Hoechst Roussel Vet, Regina, Saskatchewan), 4.2 mg, IV; cephalexin (Kefzol; Eli Lilly, Toronto, Ontario), 60 mg, IV, q8h; famotidine (Apotex, Toronto, Ontario), 2.5 mg, PO, q12h; and baytril (Enrofloxacin; Bayer, Toronto, Ontario), 10 mg, PO, q24h.

Although urine production gradually improved over the next 5 d, dyspnea developed and radiographs revealed a pleural effusion that was relieved by thoracocentesis. The radiographs also showed delayed closure of the ossification centers and scalloping of the ventral borders of the vertebral bodies (Figure 2). Hypothyroidism was suspected, based on presenting physical and clinical signs, so levothyroxine sodium (Soloxine; Daniels Pharmaceuticals, St. Petersburg, Florida, USA) was initiated at 0.025 mg, PO, q12h. Test results revealed that the total T4 was $<$ 5.15 nmol/L (reference 12.9 to 51.5 nmol L) and that the free T4 by equilibrium dialysis was $<$ 2.5 nmol/L (reference 10 to 35 nmol/L). At this point, the levothyroxine sodium was increased to 0.05 mg, q12h.

On day 14, the BUN had returned to values within normal limits and the specific gravity of the urine was 1.050.

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Figure 1. An 18-month-old intact female domestic shorthair. Note the immature kittenish appearance with small round body, short legs and neck, and broad head.

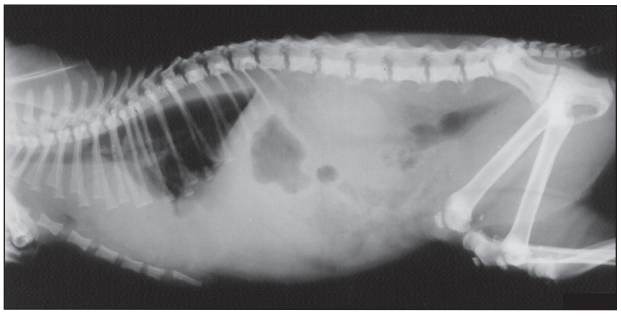

Figure 2. Radiograph prior to treatment showing delayed closure of the ossification centers. Note the scalloping of the ventral borders of the vertebral bodies.

An adrenocorticotropic hormone (ACTH) stimulation test was done to rule out hypoadrenocorticism as a potential cause for the renal failure and the possibility of multiple endocrinopathy. Test results revealed normal response to stimulation of the adrenal gland. Activity and appetite levels improved to that of a normal 1.5-year-old. The owner reported that the deciduous teeth had started to fall out and that the cat was finally beginning to show signs of estrus for the first time. One year later, the total T4 was 36.0 nmol/L (12.9 to 55.5 nmol/L) and radiographs showed that all the ossification centers had closed (Figure 3).

Naturally occurring, adult onset hypothyroidism is an extremely rare clinical disorder in cats (1). On the other hand, congenital hypothyroidism (cretinism) is also rare, but it is one of the most common causes of disproportionate dwarfism (2) and, in the literature, is better described than the adult onset form. The actual prevalence of congenital hypothyroidism may be higher than realized

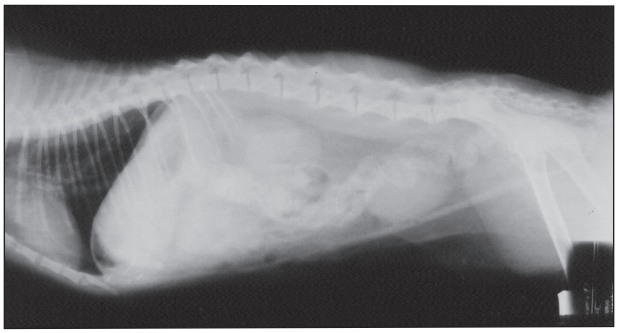

Figure 3. The same cat one year later following treatment. Note that the ossification centers have closed.

if, as is suspected, it is a potential cause of early kitten demise and thereby goes undetected (3).

Congenital hypothyroidism in cats has been reported to be caused by an iodine organification defect (2), thyroid dysgenesis (failure of the thyroid gland to form properly) (3), a defect in the anchoring of thyroid peroxidase to the cell membrane (5), or the ability of the thyroid gland to respond to thyroid-stimulating hormones (4). All reported cases of congenital hypothyroidism in cats have been due to defects of the thyroid gland. Problems arising from the pituitary or hypothalamic regions have yet to be documented.

The most obvious clinical signs of feline congenital hypothyroidism are those exhibited as disproportionate dwarfism or cretinism. These include a large broad head with short neck and limbs and a short round body. Typically, affected kittens appear normal at birth, but noticeable changes, such as a decrease in growth rate, become evident by 6 to 8 wk of age (6). As the kitten ages, more findings become evident. These include lethargy, mental dullness, constipation, hypothermia, bradycardia, prolonged retention of deciduous teeth (6), cold intolerance, and retention of the kitten hair, such that the coat remains soft and fluffy (7). Delayed closure of the ossification centers of the long bones is evident radiographically. There is also evidence of epiphyseal dysgenesis, shortened vertebral bodies, and scalloping of the ventral borders of the vertebral bodies, suggesting lack of normal longitudinal growth (6).

A presumptive diagnosis of congenital hypothyroidism can be made based on clinical signs and low basal T4 values. However, in cats, it has been documented that many concurrent nonthyroid illnesses can suppress the serum T4 concentration causing euthyroid sick syndrome (6). In this case, a free T4 assay by equilibrium dialysis is used as an adjunct diagnostic test, because it is not influenced by nonthyroid illness. As well, when a T4 concentration is low and other abnormal data support a presumptive diagnosis of hypothyroidism, a definitive diagnosis can be made after a patient successfully responds to therapy (8).

Recommended treatment for hypothyroidism is levothyroxine sodium, a synthetic form of thyroid hormone, at a starting dose of 0.05 mg to 0.1 mg, q24h. The dose can be adjusted based on clinical response and post pill serum T4 testing (3). Long-term prognosis is unknown. It has been suggested by one clinician that the reversibility of signs of congenital hypothyroidism depends on the timing of the treatment of the disease (9). Abnormal bone and joint development may cause musculoskeletal problems to persist or develop with time (6). Since thyroid hormone is necessary for adequate development of the central nervous system, treatment must be initiated within the first few weeks of life to prevent permanent mental retardation (5).

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