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Popular Article

Hypothyroidism in Canine: A Endocrinological Disorder

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Abstract

Canine hypothyroidism is most common endocrinological disorder in dogs and rare in other domestic animals. Hypothyroidism is of primary origin in about 95% of cases, affecting mainly middle-aged dogs. Labrador, Doberman, Golden Retriever, German shepherd, Spaniels, Great Dane, Dachshund have great risk. Canine hypothyroidism may occur in association with other immune-mediated endocrine disorders such as hypoadrenocorticism and diabetes mellitus. Therapeutic response has also been suggested as a method of confirming hypothyroidism in dogs with inconclusive total T4 and cTSH concentrations. levothyroxine is used for management of hypothyroidism. Regular monitoring is required and consideration needs to be given to the development of other immune mediated endocrinopathies.

Introduction

Thyroid gland located lateral to the trachea, in the region of the proximal tracheal rings. Functional unit is the thyroid follicle, which comprises the follicular cells (thyrocytes) and colloid, each gland containing both large (resting) follicles and small (active) follicles. Thyroid Hormone is a iodine-containing amino acids. The deficiency of thyroid hormone affects functions of all organs and systems, therefore results in variable clinical signs. Thyroid hormones increase the metabolic rate and oxygen consumption of most tissues, (with the exception of the adult brain, testes, uterus, lymph nodes, spleen, and anterior pituitary). Thyroid hormones are also essential for the normal growth and development of the neurologic and skeletal systems. Hypothyroidism (HpoT) is a multisystemic disease that results from deficiency of thyroid hormones (TH), thyroxine (T4) and triiodothyronine (T3) (Cooper and Ladenson 2013). Hypothyroidism is of primary origin in about 95% of cases, affecting mainly middle-aged dogs. Most frequently diagnosed endocrinopathy in dogs (Pöppl 2016) infect, one of the most over diagnosed. It is a single endocrinological disease suspected most commonly in canine suffering from alopecia (Doering and Jensen, 1973).

2. Classification of canine hypothyroidism based on etiology

(A) Primary hypothyroidism accounts for more than 95 percent of the cases and is usually caused by lymphocytic thyroiditis or idiopathic thyroid atrophy.

(B) Secondary Hypothyroidism (thyroid stimulating hormone, TSH) deficiency due to congenital malformation, pituitary destruction (tumours, trauma, autoimmune hypophysitis) or even its suppression, normally caused by hormones or drugs such as glucocorticoids (Feldman and Nelson 2015)

(C) Tertiary hypothyroidism, caused by a thyrotropin releasing hormone (TRH) deficiency due to pituitary adenoma with effacement of the overlying hypothalamus was reported.

3. Congenital hypothyroidism

Congenital hypothyroidism (CH) is one of the most common neonatal endocrine disorders, presenting with abnormal growth and intellectual impairment. Congenital hypothyroidism is caused by thyroid dysgenesis, dys-hormonogenesis, defects in the transport of thyroid hormones, TSH receptor-blocking antibodies, maternal medications, or a deficiency (endemic goitre) or excess of iodine. During the growth phase the puppy can develop hypothyroidism in the same way as the adult animal. Congenital hypothyroidism causes impaired development of the central nervous system (CNS) and skeleton. Congenital hypothyroidism is an inherited autosomal recessive trait in rat terriers, toy fox terriers, and giant schnauzers.

4. Signs of congenital hypothyroidism

Congenital hypothyroidism results in mental retardation and stunted disproportionate growth due to epiphyseal dysgenesis and delayed skeletal maturation. Vertebral physeal fracture causing tetra paresis was reported in a dog with congenital hypothyroidism. Affected dogs are mentally dull and have large, broad heads, short thick necks, short limbs, macroglossia, hypothermia, delayed dental eruption, ataxia, and abdominal distention.

5. Clinical hypothyroidism

At this stage the daily secretion of T4 is severely affected. Clinical signs vary greatly because thyroid hormone impacts myriad of systems. Clinical are not always the typical obesity, lethargy and poor hair cover as described in many textbooks (Victor, 2011).

(A) Dermatological sign

Dermatologic abnormalities are the most common presenting complaints, but they appear in later stages of the disease. Dermatologic changes occur in 60-80 percent of hypothyroid dogs. Dry scaly or greasy skin (Seborrhea sicca or oleosa), change in coloration, strong smelling of skin along with and hyperkeratosis, hyperpigmentation. Poor hair coat quality, fading of hair color, failure of hair re-growth. The hair is often brittle and easily epilated, and loss of undercoat or primary guard hairs may result in a coarse appearance or a puppy hair coat (short, softer under

coat). Myxedema (cutaneous mucinosis) is a rare dermatologic manifestation of hypothyroidism characterized by puffy but nonpitting thickening of the skin, especially of the eyelids, cheeks, and forehead and is classically referred to as 'tragic face' (Doering and Jensen, 1973). A variety of other 'atypical' and secondary dermatological abnormalities are also possible. The alopecia may be patchy and asymmetric or may only affect one area (e.g., the bridge of the nose).

(B) Neurologic dysfunction A-Peripheral neuropathy

About 2-4% dogs exhibit some behavioural changes and neurological signs such as, head tilt, seizures, ataxia, circling and facial nerve paralysis. Peripheral neuropathy caused by hypothyroidism affects primarily middle-aged and older individuals, especially of middle- to large-sized breeds.

(C) Cardiovascular system:

Sinus bradycardia, weak apex beat, low QRS voltages, and inverted T waves occur in hypothyroid dogs. Reduced left ventricular pump function but rarely myocardial failure in dogs. Dilated cardiomyopathy and hypothyroidism may occur concurrently with dramatic long-term improvement in cardiac function after treatment with l-thyroxine.

(D) Polyendocrinopathies

Canine hypothyroidism may occur in association with other immune-mediated endocrine disorders such as hypoadrenocorticism and diabetes mellitus. Hypothyroidism causes insulin resistance and may mask the classic electrolyte changes of hypoadrenocorticism.

(E) Ocular changes:

Corneal lipidosis, corneal ulceration, uveitis, lipid effusion into the aqueous humour, secondary glaucoma, lipemia retinalis, retinal detachment, and keratoconjunctivitis sicca may occurs in hypothyroidism.

(F) Haemostasis

Decreased plasma von Willebrand factor concentration has been reported in hypothyroid dogs but canine hypothyroidism is rarely associated with clinical bleeding, and platelet function.

6. Epidemiology of canine hypothyroidism

(A) Incidence

Hypothyroidism is the most common hormone imbalance of dogs with the incidence rate at about 1: 150 to 1: 500. Overall prevalence is 0.2 to 0.8 % . Central hypothyroidism reportedly accounts for less than 5% of these cases (Feldman and Nelson, 2004).

(B) Age

Dogs beyond 8 yrs. are more susceptible. Most cases are seen in dogs over one year of age middle-aged dogs and is rarely diagnosed in dogs less than two years of age (Carmel, 2003) although perhaps 10% may be in younger animals. A small number (approx. 3%) of cases are congenital, while the rest relate to pathology acquired during growth (Victor, 2011). Hypothyroidism can affect any age or breed of dog. Mean age is 7 years (range 0.5–15 years). Hypothyroidism secondary to lymphocytic thyroiditis appears to develop at a younger age than idiopathic thyroid atrophy.

(C)Breed & Sex

More common in mid to large pure-bred dogs. In india Spitz was showing high incidence (56%) followed by Labrador (28%) and German shepherd (17%). This variation could probably be due to the habitation of different breeds in different regions Spayed females and castrated males are at greater risk (Panaciera, 1994). Males and females, either neutered or entire, appear to be affected equally.

6. Diagnosis and Treatment

(A) Total triiodothyronine (T3)

T3 is three to five times more potent than T4 (pro-hormone). Measurement of T3 therefore reflects metabolic status more accurately, but it plays no role in the diagnosis of hypothyroidism, as circulating concentrations are often maintained in the reference range in hypothyroid dogs.

(B)Total-Thyroxin(T4)

Circulating total T4 concentration is invariably low in hypothyroid dogs (Dixon and Mooney, 1999). Extremely valuable screening test to rule out hypothyroidism unless anti-T4 antibodies cause a spurious increase, which are produced in 2% of dogs with hypothyroidism. Thus, a low total T4 concentration alone does not confirm hypothyroidism. Total T4 concentrations do not differ significantly between males and females but are higher in small dogs than in medium and large-breed dogs.

(C)Free thyroxin (FT4)

Free T4 is the active fraction of total T4 and is more closely reflect metabolic status at the tissue level than total T4. It is less affected by the myriad factors (drug therapies, non-thyroidal illness, breed) and T4 autoantibodies etc. capable of lowering total T4 and is thus a more specific diagnostic test for hypothyroidism. Overall, it is considered to be the best single diagnostic test for hypothyroidism but it is not without problems (Dixon and Mooney, 1999), as free T4 is only accurately measured by equilibrium dialysis or ultrafiltration techniques and these are not widely available and are relatively expensive.

(D)Endogenous thyroid-stimulating hormone (TSH)

Decreased circulating thyroid hormone concentrations reduce the negative feedback effect on the pituitary gland and consequently, in primary hypothyroidism, TSH concentrations rise. Approximately 20 to 30% of hypothyroid dogs have a TSH concentration within the reference range, which might be due to suppressive effects of concurrent non-thyroidal illnesses or from drug therapies, non-specific fluctuation, existence of secondary hypothyroidism or production of unrecognizable TSH isomers.

(E)Evaluation of the lipid profile

30-40% of all dogs with hypothyroidism present with total cholesterol elevation. It is important to measure LDL-cholesterol, since an increase in relation to HDL-cholesterol can indicate thyroid deficiency.

(D)Therapeutic response

Therapeutic response has also been suggested as a method of confirming hypothyroidism in dogs with inconclusive total T4 and cTSH concentrations. However, caution is advised. Thyroid hormone supplementation suppresses TSH production and endogenous thyroid function. Hypofunction invariably occurs once supplementation is withdrawn and can lead to confusing clinical signs for up to eight weeks later (Panciera *et al*., 1999). Thyroid hormone supplementation is known to have several physiological effects that can easily be misinterpreted as a successful response to therapy in euthyroid dogs.

(E)Thyroid Ultrasound

Ultrasound is a useful imaging tool for assessment of thyroid glands and measurement of thyroid size. The thyroid gland in many hypothyroid dogs has a smaller volume and cross-sectional area and tends to be less echogenic. Three-dimensional ultrasound is a useful and precise image method in the measurement of thyroid volume as compared with 2D ultrasonography, and this method enables to exactly detect the alteration of the thyroid lobe volume in a relatively short time.

7. How are hypothyroid dogs treated?

Synthetic T4 products have greater standardization and potency and a longer shelf life compared with crude preparations. T4 itself is considered to be a physiological pro-hormone, serves to normalize both circulating T4 and T3 concentrations and pituitary cTSH production, and can effectively be administered once daily. The recommended dose in clinical hypothyroidism is 11-22 µg/kg, starting with the lower dose and gradually increasing the dosage until the desired concentration has been reached. In adequately treated dogs, there is usually a dramatic improvement in metabolic signs within days. Dermatological abnormalities can take several months to improve and, frequently, there is worsening of alopecia before new hair re-growth

commences. There is wide inter-individual variation in gastrointestinal absorption and response to T4 replacement therapy. As a consequence, it is important to monitor dogs after commencing therapy.

8. Recommendation for treatment

Dixon and Mooney (1999) advise initially administering levothyroxine every 12h, progressing to once daily dosing). Reassessment can then be performed every 6-12 months. In the case of congenital hypothyroidism, treatment should start as soon as possible in order to avoid irreparable damage to the central nervous system. The dosage in puppies with congenital hypothyroidism or juvenile hypothyroidism is 5-20 μg/kg. Adequately treated animals have a normal life expectancy and quality of life. Regular monitoring is required and consideration needs to be given to the development of other immune mediated endocrinopathies (polyglandular syndromes). In such cases, hypothyroidism most commonly occurs in association with hypoadrenocorticism or diabetes mellitus.

9. Time of Clinical Improvement

Improvement in activity - first 1 to 2 weeks of treatment. Weight loss - within 8 weeks. Normal hair coat- several months and the coat may initially appear worse as telogen hairs are shed. Improvement in myocardial function - 8 weeks but may be delayed for as long as 12 months. Neurologic deficits - 8 to 12 weeks. Vestibular symptoms - two to four months.

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