Dietary hyperthyroidism in dogs

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**OBJECTIVES:** Evaluation of dogs with elevated plasma thyroxine concentration fed raw food before and after changing the diet.

**METHODS:** Between 2006 and 2011 all dogs presented with an elevated plasma thyroxine concentration and a dietary history of feeding raw food were included. Thyroxine (reference interval: 19·3 to 51·5 nmol/L) and in many cases also thyroid-stimulating hormone concentrations (reference interval: <0·30 ng/mL) were measured initially and after changing the diet.

**RESULTS:** Twelve dogs were presented with a median age of five years. The median plasma thyroxine concentration was 156·1 (range of 79·7 to 391·9) nmol/L; in six dogs, thyroid-stimulating hormone concentration was measured and was <0·03 ng/mL in five dogs and 0·05 ng/mL in one dog. Six dogs showed clinical signs such as weight loss, aggressiveness, tachycardia, panting and restlessness while six dogs had no clinical signs. After changing the diet eight dogs were examined: thyroxine concentration normalised in all dogs and clinical signs resolved.

**CLINICAL SIGNIFICANCE:** Dietary hyperthyroidism can be seen in dogs on a raw meat diet or fed fresh or dried gullets. Increased plasma thyroxine concentration in a dog, either with or without signs of hyperthyroidism, should prompt the veterinarian to obtain a thorough dietary history.

**INTRODUCTION**

While hypothyroidism is a common endocrine disorder in dogs, hyperthyroidism is a rare disease in this species. Although functional thyroid adenomas have been described, hyperthyroidism in dogs is almost always caused by a thyroid carcinoma (Feldman and Nelson 2004). In the 1960s, 17% of all canine malignant tumours in Germany were of thyroidal origin, whereas 50 years later only about 2% are thyroidal tumours (Kessler and Smeak 2005). Of all thyroidal tumours only approximately 10% are functional and produce thyroid hormones (Turrel and others 2006). Exogenous hyperthyroidism, a result of excessive intake of thyroid hormones, is uncommon in dogs and may result from excessive administration of sodium levothyroxine (Feldman and Nelson 2004). In human medicine, excessive consumption of meat contaminated with thyroid tissue has resulted in exogenous hyperthyroidism (Malvinder and Sturge 2003, Conrey and others 2008, Hendriks and Looij 2010) but this has so far not been reported in dogs.

Clinical signs of increased thyroxine concentration are similar to those seen in cats, such as weight loss, polyphagia, unkempt haircoat, patchy alopecia and polyuria/polydipsia (Mathes and Neiger 2010, Rijnberk and Koistra 2010).

A current trend among dog owners is the feeding of “natural” diets. Proponents argue that the processing methods used to produce commercial pet foods destroy essential nutrients and enzymes (Joffe and Schlesinger 2002). They believe that commercial pet foods do not meet the nutritional needs of dogs and may be a source of chronic health problems (Joffe and Schlesinger 2002). A natural diet widely used in Germany is commonly termed BARF (born again raw feeders, bones and raw foods, biologically appropriate raw foods). The thought behind this dietary concept is that the dog is a carnivore and therefore a perfect meal should consist of all parts of an animal with its flesh, cartilage, intestine (with ingested plant material) and bones. Because this is commonly not feasible, the basic material of this diet comes in general from various body parts of ruminants (beef, sheep, goat, deer etc) including neck with trachea and adherent thyroid gland. Thus, it is possible that a large amount of raw thyroid gland tissue ends up in the diet. These thyroid hormones are not destroyed by gastric acid and can then be absorbed, similar to levothyroxine administered for...
hypothyroidism. Elevated concentration of exogenous thyroxine may then cause clinical signs.

The sex and age, clinical signs, thyroid hormone concentration and outcome of dogs with dietary hyperthyroidism due to raw feeding are herein reported.

**MATERIALS AND METHODS**

In this retrospective study from 2006 to 2011, all dogs with increased plasma thyroxine concentration that were fed a diet of “bones and raw food (BARF)” or gullet were included. Plasma total thyroxine concentration was measured by chemiluminescence immunoassay (CLIA) (Biocontrol) with a reference interval of 19.3 to 51.5 nmol/L. In some dogs, plasma thyroid-stimulating hormone (cTSH) concentration was also measured by CLIA with a reference interval <0.30 ng/mL. With dietary hyperthyroidism as a differential diagnosis, a change of diet was proposed to the owners, either to a commercial dog food or to pure muscle meat. Re-examination between 2 weeks and 2 months after dietary change was advised to the owners.

**RESULTS**

Twelve dogs met the entry criteria for this study. Median age was five years (range 2 to 12 years) and eight were male and four were female. The median plasma thyroxine concentration was 156.1 nmol/L with a range of 79.7 to 391.9 nmol/L. In six dogs plasma cTSH was measured and was below the detection limit of the assay (<0.03 ng/mL) in five dogs and 0.05 ng/mL in one dog. Weight loss was the primary complaint in four dogs while restlessness was seen in three dogs and aggressiveness, tachycardia and panting each in one dog. Six dogs had no clinical signs and abnormally high plasma thyroxine concentrations were detected on a routine blood screen.

Dietary history showed that eight owners fed a diet of exclusively bones and raw food (BARF) while four owners fed a commercial diet but added dried gullet on an almost daily basis.

After diagnosing dietary hyperthyroidism, seven owners immediately stopped feeding dried gullet or changed from BARF to commercial dog food. Re-examination 2 weeks to 2 months later showed that plasma thyroxine concentration had decreased to below the upper limit of the reference interval in all dogs (median 36.0, range 15.4 to 42.4 nmol/L). All clinical signs of hyperthyroidism resolved within a few days after changing the diet. Repeated plasma cTSH measurement in two dogs showed continued low concentration in one and increased cTSH concentration during hypothyroidism in another (Table 1). One owner did not change the diet immediately and repeated plasma thyroxine concentration measurements continued to be elevated (one month later: >102.8 nmol/L; 4 months later: 194.0 nmol/L). The dog continued to lose weight and only when the diet was finally changed, did the clinical signs resolve and plasma thyroxine concentration returned to within the reference interval (28.3 nmol/L). Four owners did not present their dogs for a re-examination, three dogs without clinical signs and one with restlessness.

**DISCUSSION**

This retrospective study shows that dietary hyperthyroidism in dogs has to be considered when increased plasma thyroxine concentration, with or without signs of hyperthyroidism, are present.

Several possibilities can cause increased plasma thyroxine concentration. Falsely increased thyroxine concentration can be measured if the sample is stored constantly at high temperature for at least 5 days (Behrend and other 1998). It is unlikely, that this was the case in all of these dogs. While it cannot be excluded that erroneous measurement could have occurred in rare cases, increased temperature has not been shown to increased thyroxine concentration to a degree seen in the dogs of this study (Behrend and others 1998). As these animals would not show any clinical signs, this would only be likely in those dogs without clinical signs. Another potential explanation could be the occurrence of thyroid autoantibodies (T4AA). It is well known that T4AA may cause spuriously elevated plasma thyroxine concentration (Thacker and other 1992). This may have happened in some of the dogs in this study, but clinical signs of hyperthyroidism would not be expected in these cases. Furthermore, increased plasma thyroxine concentration due to T4AA occurs in only about 1% of dogs with these autoantibodies, which by themselves are seen in only 14% of sera submitted for suspected hypothyroidism (Ferguson and other 2007).

Some of the dogs in this study might have had primary hyperthyroidism due to a malignant thyroid tumour. This is unlikely because plasma thyroxine concentration normalised and clinical signs abated soon after a diet change. Furthermore, thyroid tumours commonly are large and palpable (Kessler and Smeak 2005), which was not found in any of these dogs. Rarely, ectopic or intra-thoracic thyroid tissue producing excessive thyroid hormones has been reported in dogs (Turrel and other 2006, Stassen and other 2007) but again, normalisation of plasma thyroid hormone concentration would not be expected after a diet change.

Thyrotoxicosis has been reported in dogs given excessive doses of levothyroxine for hypothyroidism (Feldman and Nelson 2004). This normally develops because of falsely calculated doses.
or communication errors with the owners. Rarely, dogs develop thyrotoxicosis when given small amounts of sodium levothyroxine (Feldman and Nelson 2004). The reason for this marked sensitivity is not known. It is unlike that the increased plasma thyroxine concentration in any of the dogs in this study was due to exogenous drug administration, as no dog was treated with these medications.

Inflammatory destruction of thyroid tissue, either immune-mediated due to a subacute de Quervain thyroiditis or non-immune-mediated due to bacterial, fungal or parasitic infection may result in increased or decreased plasma thyroxine concentration (Reed Larson and other 1998) in human patients. A similar case with temporarily increased plasma thyroxine concentration has been reported in a dog due to a badger bite (Rau and other 2007). None of the dogs in this study had clinical signs of a local infection or inflammation in the neck area. Furthermore, no therapy with antibiotics or anti-inflammatory drugs was used in any of the dogs and plasma thyroxine concentration normalised only by changing the diet.

Palpation thyroiditis has been reported in dogs following mechanical crushing (Carney and other 1975). Multifocal granulomatous folliculitis was the hallmark of the changes, but no thyroxine concentration was measured in these dogs, so it is unclear if increased, decreased or normal plasma thyroxine concentration was present. None of the dogs in this study had its thyroid glands knowingly crushed shortly before plasma thyroxine concentration measurement.

Overall, the most likely cause of the increased plasma thyroxine concentration combined with the clinical signs of thyrotoxicosis in these dogs was due to feeding thyroid tissue from animal origin. Thyrotoxicosis factitia, as it is called in human medicine, has been reported in people eating hamburgers containing ground beef thyroid or eating excessive amount of sausages containing thyroid hormones (Malvinder and Sturge 2003, Conrey and other 2008, Hendriks and Looij 2010).

It is of interest, why non-domestic animals, such as wolves or coyotes, do not suffer from thyrotoxicosis factitia. By the end of the 19th century, Cunningham performed several experiments feeding thyroid glands to animals and humans (Cunningham 1898). One of his observations was that feeding absolutely fresh and raw thyroid material (<30 minute after slaughtering) did not lead to any signs in chicken, rabbits, cats, dogs, monkeys or humans. However, when keeping the tissue on ice for a minimum of 24 hours, toxicity did develop. It is likely that the meat and gullets obtained by owners from the slaughterhouse has been cooled for a prolonged period of time, either at home or in the abattoir and thus thyrotoxicosis could have occurred.

Measurement of cTSH concentration in such patients might help to distinguish dietary hyperthyroidism from laboratory error or thyroiditis. Although current cTSH assays cannot be used to document suppressed TSH because of poor assay sensitivity, values below the detection limit – as seen in this study – are highly suggestive of increased endogenous (thyroid tumour) or exogenous thyroxine concentration (dietary hyperthyroidism).

In conclusion, dietary hyperthyroidism can be seen in dogs fed a raw meat diet or fresh or dried gullets. Increased plasma thyroxine concentration in a dog, irrespective if signs of hyperthyroidism are present, should prompt the veterinarian to obtain a thorough dietary history. Pet owners should be advised that feeding these diets might result in dietary thyrotoxicosis.

Conflict of interest
None of the authors of this article has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

References