Case series

Thyrotoxicosis in dogs caused by consumption of thyroid-contaminated ground beef

Introduction

canine hyperthyroidism is exceptionally rare and typically results from thyroid carcinomas producing excessive amounts of thyroid hormones (Mooney and Jones, 2004). Sporadic cases associated with a functional adenoma have been reported (Lawrence et al., 1991, Itoh et al., 2007). Elevations of thyroid hormones are usually modest and clinical signs are mild (Meuten, 2002; Graham and Mooney, 2005). Other published causes of high T4 measurements include anti-thyroxine (T4) antibodies interfering in the immunoassay (Rajatanavin et al., 1989; Choi et al., 2006), prolonged storage of serum at high temperatures (Behrend et al., 1998), thyroid trauma (Rau et al., 2007), discontinuation of the administration of substances blocking T4 synthesis (Frank et al., 2005; Sellig et al., 2008) and levothyroxine treatment (Feldman and Nelson 2004, Fine et al., 2010). Recently Köhler et al. (2012) described a new cause of hyperthyroxinemia in 12 dogs fed with bones and raw food diet (BARF). Six of these dogs showed clinical signs of thyrotoxicosis including weight loss, restlessness, aggressiveness, tachycardia and panting that disappeared after changing the diet. The authors speculated that the thyrotoxicosis was most likely caused by feeding thyroid tissue from animal origin.

The present paper describes the simultaneous occurrence of hyperthyroxinemia in 6 dogs caused by the consumption of raw ground beef demonstrably contaminated with thyroid gland tissue.

Animals, Materials and Methods

Two female spayed Rhodesian Ridgeback dogs with 11 and 13 years of age were admitted to the local clinic because of laboratory values compatible with hyperthyroidism (Fig 1, dog 1 and 2). The dogs, living in the same house-hold, had a history of gradually worsening polydipsia (4-6 L/24 hours each)/polyuria, excessive panting and restlessness. Appetite was good and weight loss had not been observed. Recently one of the dogs had collapsed during a walk and despite intensive examinations at a local clinic including cardiac and abdominal ultrasonography, no underlying disease had been identified. Sinus tachycardia was the only abnormality detected. One year earlier the owner had started with a raw food diet including ~ 300-500 grams of ground beef/day. Meat was stored in the deep freezer.

Both dogs were in good physical condition and clinical examinations including palpation of the neck area were unremarkable. Ultrasonographic evaluation of the ventral aspect of the neck revealed normal shaped but small thyroid glands (total volume 485 and 235 mm3; Brömel et al., 2006). A 1 x1 cm cyst was detected in the cranial part of the left thyroid gland in the older dog. Results of a complete blood count were unremarkable. Plasma biochemical analysis including glucose, creatinine, urea, cystatin C, alkaline phosphatase, calcium, sodium, potassium, triglycerides, cholesterol and creatinine kinase were within the reference values. Alanine amino transferase (168 U/l, norm. <55 U/l) and aspartate aminotransferase (36 U/l, norm. < 25 U/l) were slightly elevated in the younger dog. Urine was isosthenuric and contained no active sediment. Urinary protein to creatinine ratio was measured in the older dog and was 0.02 (norm< 0.5). Thyroglobulin, T3 and T4-antibodies measured with an enzyme immunoassay (Patzl and Möstl, 2003) were negative.

The simultaneous occurrence of functional ectopic thyroid neoplasia in two dogs of the same owner was considered very unlikely. As the dogs had no access to thyroid medication, a causative relationship with the feeding protocol was taken into account. The hypothesis was that the ground beef had been contaminated with bovine thyroid gland. To substantiate this hypothesis the slaughtering plant was consulted, iodine concentration of the ground beef was analyzed, T4, and TSH concentrations were measured in 5 other dogs receiving the same diet and hormone concentrations were monitored after reducing or eliminating the allotment of the specific raw beef from the diet.

The butcher confirmed that meat had been harvested from the neck and that the thyroid glands had not been removed. The iodine concentrations measured by inductively coupled plasma mass spectrometry (ICP-MS) in two random samples collected before and after removal of thyroid tissue were 9.43 ± 2.36 mg/kg (~ 15 mg/kg dry matter) and < 0.08 mg/kg, respectively (average iodine content: meat ~ 0.02-0. 15 mg/kg; thyroid gland ~378-844 mg/kg, Flachowsky et al., 2007). From the iodine content it was concluded that the meat had indeed been contaminated with thyroid tissue. Daily iodine intake of the two dogs was at least 247µg/kg 0.75 compared to the recommended allowance of 29.6µg/kg0.75 (NRC 2006). Remarkably, four of the five control dogs fed with beef from the same slaughter plant showed hyperthyroxinemia as well (Fig 1). Two dogs had changed behavior and were restless and aggressive. In all seven dogs T4 concentrations declined after changing the diet (T4 [median and range]: first measurement: 86.2 nmol/l [47.6 to 193.1], control 1: 17.6nmol/l [below detection limit given as 6.3 to 35.6], control 2: 15.4 nmol/l [6.3 to 20.6 nmol/l]; Wilcoxon test (first measurement vs. control 1 and 2: p = 0.018; control 1 vs. control 2: p = 0.499). Thyroid stimulating hormone stayed undetectable to low throughout the observation period.

Discussion

This is the second report of food induced thyrotoxicosis in dogs. Thyroid hormones are orally active and feeding fresh thyroid glands was one of the first effective treatments of hypothyroidism in human patients (Mackkenzie, 1892). Thus it is not surprising that the consumption of thyroid contaminated food can cause exogenous hyperthyroidism. In fact, two large outbreaks of thyrotoxicosis later called “hamburger thyrotoxicosis” in the United States in 1984 and 1985 with a total of 170 patients (Hedberg et al., 1987; Kinney et al., 1988) resulted in the prohibition of gullet trimming by the United States Department of Agriculture. Gullet trimming is a method that removes the muscles from bovine larynx for ground beef. Since then sporadic cases from Canada (Parmar and Sturge, 2003) and the Netherlands (Hendriks and Looij, 2010) have been reported. Recently 12 dogs with food associated hyperthyroxinaemia were identified in a retrospective study in Germany from 2006 to 2011 by Köhler et al. (2012). Clinical signs included weight loss, tachycardia, excessive panting, aggressiveness and restlessness. Except for weight loss, these signs were also observed in the dogs of the present study and are in accordance with observations in dogs with functional thyroid neoplasia or excessive administration of levothyroxine (Feldman and Nelson, 2004). Polydipsia/polyuria, the most consistent clinical findings in dogs with functional thyroid tumors (Meuten 2002) were observed in two of six dogs with hyperthyroxinemia in this study. Possible causes of collaps, as reported in one of the dogs include cardiovascular manifestations of thyrotoxicosis (Fine et al., 2010) and thyrotoxic hypokalemic paralysis (Kung 2006). The latter is a common complication of hyperthyroidism in human patients, but has not been described in dogs.

Whether thyrotoxicosis was caused by excessive iodine or T4 in the diet was not determined. In human patients iodine induced hyperthyroidism is sometimes observed in susceptible individuals with preexisting thyroid disease including iodine deficiency goiter (Roti and Uberti 2001). The facts that the dogs had atrophic thyroid glands (Thompson 1979), were fed with commercial dog food before changing to raw food diet and iodine excess normally leads to hypothyroidism through the Wolff-Chaikkoff effect (Castillo et al., 2001) supports the hypothesis that thyrotoxicosis was caused by the ingestion of T4.

In conclusion, the work-up of dogs with hyperthyroxinemia should include a thorough investigation of feeding habits, as thyrotoxicosis can be caused by the consumption of thyroid-contaminated beef.

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