Metabolic bone disease and hyperparathyroidism in an adult dog fed an unbalanced homemade diet

Metabole botafwijkingen en hyperparathyroïdie bij een volwassen hond na consumptie van een slecht uitgebalanceerd huishouddieet

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ABSTRACT

An 8-year-old intact male Briard was presented with a non-painful bilateral diffuse swelling of mandible and maxilla. The teeth were mobile. The jaws felt like rubber. Radiographic examination revealed generalized osteopenia. Ultrasound showed prominent parathyroid glands. The plasma parathyroid hormone concentration was extremely high, the serum 25-hydroxy-vitamin D (25-OH Vit D) was low, and the serum ionized calcium remained within the reference range. As the dog had been fed an unbalanced homemade diet for many years, rubber jaw, osteomalacia and secondary hyperparathyroidism due to dietary calcium and vitamin D deficiency were diagnosed. Dietary correction resulted in clinical improvement and normalization of the plasma parathyroid hormone concentration within 4 months. However, although the 25-OH Vit D was clearly raised, it still did not reach reference values, which was due to lack of owner compliance, as the owner had changed the patient's diet over time.

SAMENVATTING

Een acht jaar oude, mannelijk intacte briard werd aangeboden met een niet-pijnlijke diffuse zwelling van de manibula en maxilla. De tanden stonden los. De kaken voelden aan als rubber. Radiografisch onderzoek toonde een algemene osteopenie. Ultrasonografie liet een prominente zwelling van de bijschildklieren zien. De plasma parathyroïd hormoonconcentratie was extreem hoog, de serum 25-hydroxy-vitamin D (25-OH Vit D) concentratie was laag en het serumgeïoniseerd calcium bleef binnen de referentiewaarden. Aangezien de hond ook reeds vele jaren een niet-uitgebalanceerd huishouddieet als voeder kreeg, werd de diagnose van *rubber jaw*, osteomalacie en secundaire hyperparathyroïdie ten gevolge van een diëtair calcium- en vitamine D-tekort gesteld. Na vier maanden resulteerde de correctie van het dieet in een klinische verbetering en een normalisatie van de plasma parathyroïd hormoonconcentratie. Hoewel de serum 25-OH Vit D-waarde reeds een duidelijke stijging vertoonde, werden de referentiewaarden niet bereikt. Dit kwam omdat de eigenaar het dieet van de patiënt na verloop van tijd veranderde.

INTRODUCTION

In developed countries, more than 90% of dogs and cats consume complete and balanced commercially prepared pet foods for at least half of their diet (Laflamme *et al*., 2008). However, the use of non-commercial diets, including homemade and raw food diets, has risen in popularity among veterinarians and pet owners. For 30.6% of dogs and 13.1% of cats, table scraps, leftovers and homemade foods are fed as part of the main diet. Currently, 3% of dog and cat owners feed their pets exclusively with homemade foods (Laflamme *et al*., 2008). When properly formulated and prepared, these diets can provide complete and balanced nutrition. However, dogs fed homemade diets perceived by the owners to be complete had a greater prevalence of health problems compared to dogs fed nutritionally balanced commercial foods, because there is a greater potential for nutrient deficiencies, excesses and imbalances with homemade diets (Rahman and Yathiraj, 2000).

Nutritional secondary hyperparathyroidism (NSHP), osteodystrophy and osteopenia are common complications that can develop in pets consuming imbalanced diets. The basic underlying cause of NSHP is calcium deficiency, which can occur due to inability to absorb Ca, lack of dietary Ca and/or vitamin D (Vit D), or excessive dietary phosphorus, even when the Ca intake is adequate (Miller, 1969; Bennett, 1976). Due to the high

Ca requirement, growing and lactating pets are found to be especially susceptible. Several cases of NSHP have been reported in puppies (Saville *et al*., 1969; Lourens, 1980; Kawaguchi *et al*., 1993; Valat and Asimus, 2000; Taylor *et al*., 2009), but to date and to our knowledge, only one case study of NSHP has previously been reported in an adult dog (de Fomel-Thibaud *et al.*, 2007).

This report primarily describes the diagnosis of NSHP, osteomalacia and rubber jaw in an 8-year old dog, based on clinical findings, on parathyroid hormone (PTH) and 25-hydroxycholecalciferol (25-OH Vit D) assays, and on ultrasound and radiographic studies. The dietary treatment of the patient is also discussed.

CASE REPORT

An 8-year and 9-month-old, intact male Briard was presented with a history of weight loss, inappetence, weakness and depression. For the previous four months, the dog had shown excessive salivation and the owner noticed thickening of the dog's gingiva. The dog had difficulty chewing and preferred soft food. One month earlier, the dog had become rather immobile, developed lameness on its front limbs and became unable to perform any activity. The dog had been fed an unbalanced homemade diet for many years because it easily developed diarrhea with any kind of commercial dog food. Throughout the dog's lifetime, the owner had tried many different commercial diets, as well as many different protein sources when preparing the homemade diet. Intermittent episodes of diarrhea occurred when the dog was fed beef, veal, pork, lamb, chicken and turkey, whereas the feeding of horse meat did not evoke diarrhea. To date, no elimination-challenge trial had been performed in order to diagnose adverse reactions to food. At the time of the consultation, the dog showed no diarrhea and was receiving a pasta and horse meat-based diet. The dog's daily diet consisted of 100 g horse meat (uncooked weight, UW), 120 g pasta (UW), 400 g bread (variable based on what was available), 500 g carrots (UW), 50 g spinach, one (100 g) tomato and two teaspoons (15 g) of coconut oil. The horse meat, pasta, carrots and spinach were cooked and mixed with the other ingredients. Neither vitamin nor mineral supplements were provided.

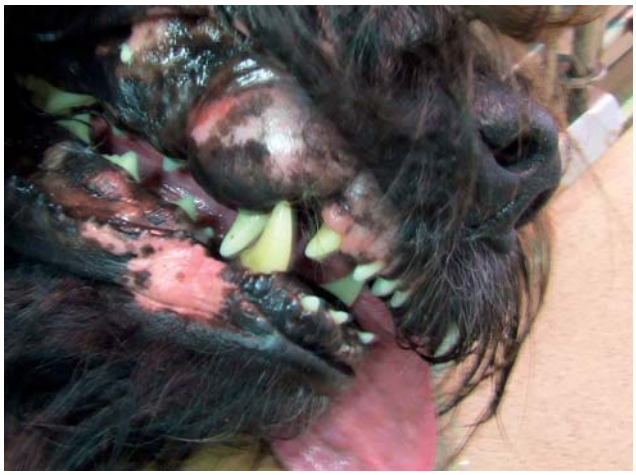

Figure 1. Marked diffuse thickening of mandible and maxilla in a dog with rubber jaw due to secondary nutritional hyperparathyroidism.

Physical examination revealed an underweight body condition: body weight 38 kg; ideal body weight 42 kg, body condition score 3 to 9 according to a nine-point scale (Laflamme, 1997). The dog was very calm and listless, it preferred to lie down, and it had difficulties getting up, standing and walking. Moderate muscle atrophy of the front and hind limbs was also noticed. Abdominal palpation and neurological examination showed no abnormalities. Examination of the oral cavity showed marked diffuse thickening of both jaws without ulceration (Figure 1). Palpation revealed non-painful bilateral swelling of the mandible and maxilla. The jaws felt like rubber and could have been crushed with digital pressure. The teeth were mobile without pocketing.

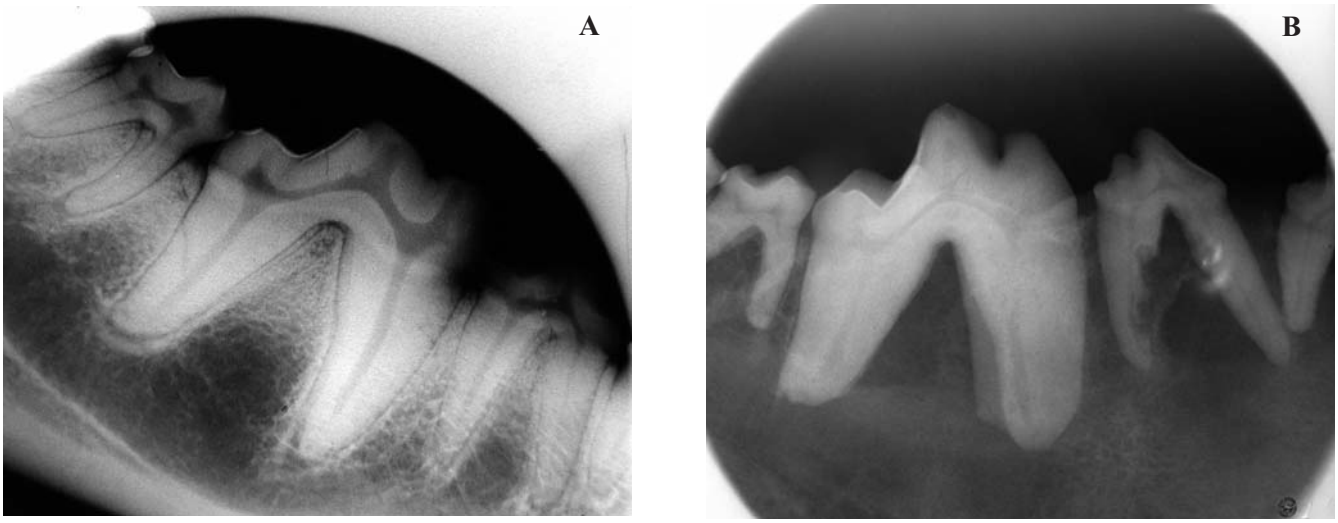

Figure 2. Radiographic lateral projections of the mandible of a healthy dog (A) compared to the mandible of the dog diagnosed with rubber jaw due to secondary nutritional hyperparathyroidism (B). In this case of rubber jaw (B), the bone opacity is reduced, the trabeculae are no longer visible and the laminae dure dentes have disappeared, so that it seems like the teeth are floating in soft tissue.

Table 1. Initial blood work and urinalysis in a dog diagnosed with rubber jaw following secondary nutritional hyperparathyroidism, before and 4 months after starting treatment.

	Pre-treatment	4 Months after starting treatment	Reference range*
Routine blood analysis:			
Serum biochemistry			
Total Ca (mmol/l)	2.28	2.38	2.21 – 2.79
P (mmol/l)	0.87	0.94	0.90 – 1.77
BUN (mmol/l)	5.49	7.33	1.66 – 8.65
Creatinine (µmol/l)	46.9	65.4	<100
Total protein (g/l)	54	55	54 – 76
AST (U/l)	75	21	<44
ALT (U/l)	124	42	<52
GGT (U/l)	41	<3	<8
ALP (U/l)	281	57	<123
ALP after inactivation (U/l)	<1	-	
Glucose (mmol/l)	5.27	4.16	3.05 – 4.99
Urinalysis			
White blood cells/µl	28	16	<25
Red blood cells/µl	919	9	<25
pH	7.5	6.5	4.5 – 7.0
Urinary specific gravity	1025	1041	1015 – 1035
Protein:creatinine ratio	9.27	1.6	<0.5
Particular hormonal analyses			
iCa (mmol/l)	1.27	1.45	1.25 – 1.45
PTH (pmol/l)	>210	4.6	3.0 – 17.0
PTHrP (pmol/l)	0.0	0.0	0.0
25-OH Vit D (nmol/l)	4	45	60 – 215

Total Ca, serum total calcium concentration; P, serum phosphorus concentration; BUN, blood urea nitrogen concentration; AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, γ-glutamyltransferase; ALP, alkaline phosphatase; iCa, serum ionized calcium concentration; PTH, plasma parathyroid hormone concentration; PTHrP, plasma parathyroid hormone reactive protein concentration; 25-OH Vit D, serum 25-hydroxyvitamin D concentration.
*Reference ranges provided by the external laboratories. All parameters were analyzed at Algemeen Medisch Labo (AML), Antwerp, Belgium, except for iCa, PTH, PTHrP, and 25-OH Vit D, which were determined at the Diagnostic Center for Population and Animal Health at Michigan State University, Lansing, MI, USA.

Complete blood count and serum biochemistry were unremarkable, except for increased serum γ-glutaryltransferase (GGT) and alkaline phosphatase (ALP) activities. The increased serum aspartate aminotransferase and alanine aminotransferase activities were not significant (Table 1). Urinalysis revealed marked proteinuria. Because cytological examination of the urinary sediment showed large numbers of spermatozoids and erythrocytes, the proteinuria was considered to be of postrenal origin. Radiographic examination of the teeth (Figure 2) and skull bones, showed severe reduction in radiopacity corresponding with severe osteopenia. There was thinning of the cortices of the mandibula, and the laminae dura dentes had disappeared. Therefore, it seemed like the teeth were floating in soft tissue. The severe osteopenia was indicative of metabolic bone disease for which NSHP, rickets/osteomalacia, primary hyperparathyroidism, humoral hypercalcemia of malignancy, renal osteodystrophy and hypervitaminosis A are important differential diagnoses (Johnson and Watson, 2000). In view of the age of the dog and the fact that humoral hypercalcemia of malignancy could be a possible underlying cause for the osteopenia, thoracic radiographs were taken to check for the presence of neoplasia. No abnormalities were detected, except for reduction in radiopacity of the scapula and bone structures surrounding the thorax. An abdominal ultrasound was performed because of the long history of intermittent diarrhea and to evaluate the kidneys. No significant abnormalities were detected, except for the heterogeneous appearance and enlargement of the prostate. Ultrasound-guided fine needle aspiration of the prostate was consistent with benign prostatic hyperplasia. An ultrasound of the neck was performed to evaluate the parathyroid glands (Figure 3). All four parathyroid glands were ovoid and hypoechoic in relation to the surrounding thyroid parenchyma. Compared to normal values (diameter: < 2 mm: length: < 3.3 mm) (Reusch *et al.*, 2000; Wisner *et al.*, 2009), they were all increased in size. The generalized osteopenia, the elevated serum GGT and ALP activities (which might have been due to increased osteolytic activity and bone resorption), and the enlargement of all the parathyroid glands were indicative for hyperparathyroidism, which can be primary (Berger and

Table 2. Estimated nutrient composition of the different diets fed prior to treatment and during treatment of a dog diagnosed with rubber jaw following secondary nutritional hyperparathyroidism; calculated by use of nutrient composition tables (Nubel, 2009).

	NRC[1]	Pre-treatment	Treatment			Follow-up
		Diet A	Diet B	Diet C	Diet D	Diet E
Ingredients (g/d)						
Horse meat (UW)		100	-	125	600	400
Pasta (UW)		120	-	50	200	100
Bread		400	-	-	-	400
Coconut oil		15	-	-	-	-
Canola oil		-	-	-	10	-
Carrots (UW)		500	-	250	200	400
Spinach		50	-	-	-	-
Tomato		100	-	-	-	-
Thyme		+	-	-	-	-
Bay leaves		+	-	-	-	-
Garlic		+	-	-	-	-
Hill's Feline Z/D® ULTRA allergen-free canned[2]		-	1495	1015	-	-
Calcium carbonate		-	-	6.5	-	-
Vitamin-mineral premix[3]		-	-	-	9	6
Amount fed (g)	NA	1278	1495	1446	1069	1306
ME (kJ/100g)	NA	1831	1636	1479	1453	1940
Energy intake ($kJkg^{0.75}$)	418	464	414	377	368	494
Per 100 g DM						
Crude protein (g)	10	16.1	33.7	33.2	41.3	26.7
Crude fat (g)	5.5 [33]	5.1	18.2	13.9	6.3	4.0
NFE (g)	NA	71.9	40.2	43.0	43.4	62.0
Crude fibre (g)	NA	5.6	1.5	3.7	3.9	4.0
Crude ash (g)	NA	2.8	6.4	7.2	6.0	4.3
Ca (g)	0.4	0.07	0.72	1.23	0.60	0.32
P (g)	0.3	0.20	0.64	0.57	0.54	0.35
Ca:P	1 – 0.5	0.35	1.12	2.16	1.11	0.92
K (g)	0.4	0.58	0.80	0.84	0.75	0.58
Na (g)	0.08 [>1.5]	0.60	0.30	0.26	0.16	0.60
Vit D (IU)	55 [320]	0.00	132.58	95.35	81.62	40.24

UW, uncooked weight; ME, metabolizable energy; DM, dry mater; NFE, nitrogen-free extract; Ca:P, calcium to phosphorus ratio; Vit D, vitamin D; NA, not applicable.
[1]NRC (2006b): Recommended nutrient allowances and safe upper limits for maintenance of adult dogs.
[2]Ingredient list of the commercial diet[e]: Hill's Prescription Diet® Feline Z/D® ULTRA allergen-free, Canned: hydrolyzed chicken liver, corn starch, vegetal oil, cellulose, calcium carbonate, DL-methionin, dicalcium phosphate, potassium chloride, iodinated salt, taurine, calcium sulphate, vitamins and trace elements.
[3]Vitamin-mineral premix consisting of Ca 22.2%, P 5.0%, Na 1.7%, Mg 2.2%, I 0.006%, Cu 0.022%; Mn 0.024%, Zn 0.18%, Cl 2.6%, VitA 2888,9IU/g, Vit D 33.33 IU/g, VitE 2.222 mg/g, VitB1 0.133 mg/g, VitB2 0.311 mg/g, VitB6 0.089 mg/g, VitB12 2.11 µg/g.

Feldman, 1987; Mellanby and Herrtage, 2004) or secondary to renal disease (Nielsen, 1954; Kyle *et al*., 1985) or can follow prolonged intake of an incomplete and unbalanced diet (Miller, 1969; Bennett, 1976). To differentiate between these causes, plasma and serum were collected and frozen for determination of PTH, parathyroid hormone related protein (PTHrP), 25-OH Vit D and ionized calcium (iCa) (Table 1). Intact PTH as well as PTHrP were analyzed by a two-site immunoradiometric assay (DSL-8000 (intact PTH) and DSL-8100 (PTHrP), Diagnostic Systems Laboratories Inc., Webster, TX, USA). 25-OH Vit D was measured using a commercial radioimmunoassay (25-hydroxyvitamin D ^{125}I RIA Kit; DiaSorin, Stillwater, MN, USA) and iCa was determined using an ion-sensitive electrode (Nova 8+ Electrolyte Analyzer, Nova Biochemical, Waltham, MA, USA). Plasma PTH concentration was extremely elevated, serum 25-OH Vit D concentration was dec-

reased, serum iCa concentration was within the reference range and PTHrP was undetectable.

Normal values of total Ca and iCa ruled out primary hyperparathyroidism and humoral hypercalcemia of malignancy. Furthermore, the absence of PTHrP made humoral hypercalcemia of malignancy also unlikely. Therefore, secondary hyperparathyroidism was diagnosed. Based on the absence of azotemia or isosthenuria and based on the normal appearance of the kidneys on ultrasonography, renal failure was ruled out as a possible cause. Hence, the secondary hyperparathyroidism was most probably associated with prolonged feeding of a nutritionally incomplete and unbalanced diet.

The nutritional composition of the dog's diet (Table 2, Diet A) was estimated by calculations using composition tables (Nubel, 2009), which revealed a more than sufficient energy intake (464 kJ/kg$^{0.75}$) compared to the maintenance energy requirement of older dogs established by the National Research Council (NRC) (2006a). Nevertheless, there was found to be an imbalance of macronutrients. As coconut oil was the only vegetable source of fat, essential fatty acid deficiency was also suspected. Furthermore, this diet led to Ca, P and Vit D deficiency because it contained 0.07% Ca and 0.20% P DM, it had a Ca:P ratio of 1:2.8 and it provided only limited amounts of Vit D. This incomplete and unbalanced diet confirmed secondary nutritional hyperparathyroidism, rubber jaw and osteomalacia.

The treatment consisted mainly of dietary correction of the ration by providing a balanced commercial diet. Castration was recommended because of the benign prostate hyperplasia, and endoscopic evaluation of the gastrointestinal tract was advised if any further episodes of diarrhea should occur. In view of the history of diarrhea, the thin body condition and the inability to chew, a hypoallergenic canned cat food (Hill's Prescription Diet® Feline Z/D® ULTRA allergen-free, Canned, Hill's Pet Nutrition, Topeka, Kansas, USA) was advised, as cat foods generally contain higher amounts of protein compared to the commercially available hypoallergenic canned dog foods. The amount of food was calculated to fulfill the energy requirement for older dogs established by the NRC (2006a) in order to maintain an ideal body weight of 42 kg. The nutrient composition of this diet is shown in Table 2, Diet B. The diet contained nutritionally adequate but not excessive amounts of Ca, P and Vit D, in accordance with the NRC recommendation (2006b). The Ca:P ratio was also according to the NRC recommendations (2006b). As this diet contained a much higher amount of crude fat, the owner was advised to change the diet gradually over seven days, but instead she switched to the commercial cat food at once, and this resulted in a new episode of diarrhea. As the owner was convinced that the commercial diet was the cause of the diarrhea and was therefore reluctant to continue it, the decision was taken to formulate a combination of the commercial and the homemade diet, consisting of 1015 g Hill's Feline Z/D, 125 g horse meat (UW), 50 g pasta (UW),

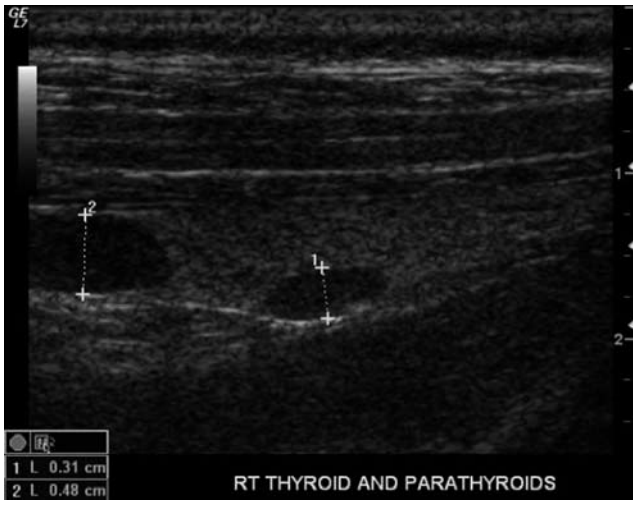

Figure 3. Ultrasound of the neck of the dog. All four very prominent parathyroid glands were ovoid and hypoechoic in relation to the surrounding thyroid parenchyma. On the right side, the cranial parathyroid gland measured 4.8 mm in height and 9.1 mm in length, and the caudal parathyroid gland measured 3.1 mm and 7.0 mm, respectively.

and 250 g carrots (UW). In order to achieve a Ca:P ratio of 2:1, 6.5 g calcium carbonate was supplemented. Dietary Vit D (95 IU/100g DM) also remained within the NRC recommendations (2006b). The nutrient composition of this diet is also shown in Table 2, as Diet C. With this diet, however, the dog continued to have episodes of diarrhea. Nonetheless, after one month the dog was markedly better. It had a good appetite, gained 3 kg in the one month (body weight 41 kg), was able to go for long walks, and dared to carry a stick in his mouth. Little by little, the patient tried to chew, though this remained a difficult task. Since the swelling of the jaws had already slightly diminished, the owner was advised to continue with the prescribed combination diet. One month later, the owner requested to switch the diet to a balanced homemade diet, without any addition of commercial food, because the owner claimed that the dog was still having episodes of diarrhea. Therefore, a homemade diet consisting of 600 g horse meat (UW), 200 g pasta (UW), 10 g canola oil and 200 g carrots (UW) was formulated as shown in Table 2, Diet D. Because of recurrent diarrhea, the fat content of the diet was also reduced. A tailor-made vitamin and mineral premix (9 g daily) was added to balance the diet. The Ca:P ratio of this diet was 1.1:1, and the Vit D content was 82 IU/100g DM. Four months after the first consultation, the dog reached its ideal body weight. The jaws were still swollen, but felt firm. The teeth were immobile and the dog was able to chew. Blood work and urinalysis were repeated (Table 1). PTH was markedly decreased and remained within the reference range, whereas 25-OH Vit D was clearly raised, though it still did not reach reference values. The owner admitted that she had discontinued the vitamin and mineral premix as well as the canola oil from the diet for the

past three weeks, as she thought these ingredients were the cause of the new episode of diarrhea. Whenever a new episode occurred, the patient was given antibiotics (trimetoprim-sulfa, Tribrissen®, Shering-Plough n.v., Heist-op-den-Berg, Belgium), following the advice of the referring veterinarian, and the diarrhea responded well. Because of the recurrent small and large bowel diarrhea, a gastroduodenoscopy and colonoscopy were performed and biopsies were taken. Histopathologic examination of stomach biopsies showed very extensive lymphoid aggregates and marked fibrosis in the lamina propria, along with several subepithelial capillary hemorrhages and glandular atrophy in the antrum. Giemsa staining showed substantial amounts of mast cells but failed to reveal any evidence of bacteria. Duodenal biopsies revealed short and stump villi, villus fusion and dilatation of centrolobular lacteals. The lamina propria of the duodenum showed mild to moderate infiltration of lymphocytes and plasma cells, whereas the lamina propria of the ileum showed dilatation of central lacteals and infiltration of eosinophilic granulocytes. Finally, colonic biopsies showed several small capillary hemorrhages, mild multifocal plasma cell infiltrate in the lamina propria, and random spread infiltration of eosinophilic granulocytes. Giemsa staining also showed a marked increase in activated mast cells in the lamina propria. The final histological diagnosis was allergy-mediated enteritis, which could not be classified in the standard entities. The owner was again advised to strictly follow the nutritional recommendations. At that time, corticosteroids could not be prescribed, as the NSHP and rubber jaw had not yet completely been resolved and the corticosteroids had further reduced the blood calcium levels. However, 1.5 years after first presentation, the patient is still recovering well from rubber jaw and the owner is no longer complaining about diarrhea. The patient has also gained another 6 kg (current body weight 48 kg), most probably because the energy intake was increased (494 kJ/kg$^{0.75}$) when the owner adjusted the prescribed homemade diet. The following changes were made by the owner: 400 g horse meat (UW) instead of 600 g, 100 g pasta (UW) instead of 200g, 400 g carrots (UW) instead of 200 g, and 400 g of bread per day was added to the diet. The administration of canola oil was again terminated and the amount of vitamin and mineral premix was decreased from 9 g to 6 g. These changes resulted not only in increased energy intake, but also in the Ca, P and Vit D intakes being decreased, even to levels below the NRC recommendations (Table 2, Diet E). As the dog had no major problems and the CBC and serum biochemistry were unremarkable at that moment (data not shown), it was impossible to convince the owner to follow earlier dietary recommendations. PTH and 25-OH Vit D analyses were not repeated at that time.

DISCUSSION

In this dog, severe reduction of the bone mass of the skull bones, scapula and bone structures surrounding the thorax had occurred, which were indicative of metabolic bone disease. Based on the dietary history, clinical findings, blood and urinalysis, hormone measurements, radiographic examination and ultrasound of neck and abdomen, this dog was diagnosed with NSHP. However, in the present case, a combination of Ca and Vit D deficiency was reported, rather than an isolated Vit D or Ca deficiency.

Isolated Ca deficiency or excess P, either of which can induce hypocalcemia, are the most common causes of NSHP (Miller, 1969; Bennett, 1976; Johnson and Watson, 2000). Hypocalcemia stimulates the parathyroid glands, which leads to hyperplasia and enlargement of these glands, as observed in this patient (Bennett, 1976). Hypocalcemia also leads to increased PTH secretion by these glands, which normalizes the blood Ca concentration by promoting mineral resorption from bone (Miller, 1969; Bennett, 1976; Saville *et al.*, 1969). Chronic ingestion of an imbalanced diet maintains the hyperparathyroid state, thus resulting in progressive skeletal demineralization and consequent clinical signs. Clinically, NSHP is primarily a problem in young growing dogs and in lactating dogs, because of the high Ca requirements of both groups (Bennett, 1976; Lourens, 1980; Kawaguchi *et al*., 1993). Young animals exhibit the typical pathology of the long bones and spine, such as swollen costochondral junctions and metaphyses, spontaneous fractures of long bones, limb deformation, vertebral compression and pelvic collapse. The effects are less dramatic in adults, mainly involving the skull bones and the rubber jaw syndrome. This syndrome is clinically characterized by swollen and pliable maxilla and mandibula, and by misaligned, loose or lost teeth (Johnson and Watson, 2000). The clinical presentation of the present patient also included rubber jaw syndrome. This syndrome has also been observed in a small number of dogs with renal osteodystrophy (Nielsen and McSherry, 1954; Kyle *et al.*, 1985) and only rarely in dogs with primary hyperparathyroidism (Berger and Feldman, 1987; Mellanby and Herrtage, 2004). To date and to our knowledge, only one other case study has reported rubber jaw due to Ca and Vit D deficiency following chronic ingestion of an imbalanced homemade diet (de Fomel-Thibaud, 2007).

As seen in the present study, nutritional history is very important for the diagnosis of NSHP, whereas blood biochemical analyses are of little value in confirming this disorder. Usually, serum Ca concentration is normal because of compensatory changes (Bennett, 1976). This is in contrast to primary hyperparathyroidism and humoral hypercalcemia of malignancy. Furthermore, ALP and GGT may appear high due to increased osteolytic activity and bone resorption, however, this finding is non-specific (Lourens, 1980). Still, blood biochemical tests as well as urinalysis and abdominal ultrasound are necessary to differentiate between NSHP and renal osteodystrophy. Hormone measurements are especially important for

confirming NSHP (Rosol and Capen, 1996). In the present study, PTHrP was not detected, which makes humoral hypercalcemia of malignancy unlikely (Rosol and Capen, 1996), whereas the PTH concentration was extremely high pre-treatment but returned to reference values post-treatment.

Increased PTH initiates conversion of 25-OH Vit D to calcitriol (1,25-OH Vit D) in the kidneys, which then stimulates intestinal Ca absorption (Rosol and Capen, 1996). Therefore, in the absence of Vit D deficiency, the increased PTH would have been associated with a mild increase in serum 25-OH Vit D concentration. Nonetheless, an extremely low serum 25-OH Vit D concentration was observed in this patient. As dogs do not synthesize cholecalciferol adequately in the skin when radiated with ultraviolet light, they mainly depend on the dietary intake of Vit D (How *et al.*, 1994). Accordingly, calculation of the dietary Vit D intake also confirmed the occurrence of hypovitaminosis D. Even so, intestinal problems, such as inflammatory bowel disease, may worsen the development of hypocalcemia secondary to diminished intestinal absorption of Vit D (Mellanby and Herrtage, 2004; Kimmel *et al.*, 2000). And, as fat-soluble vitamins are absorbed along with dietary fat and are often associated with these lipids, any disturbance of normal lipid absorption negatively influences the uptake of fat-soluble vitamins from the intestine (NRC, 2006c). Therefore, Vit D deficiency might also result from limited fat intake. Nevertheless, in this patient the extremely low dietary intake remains the most important reason. Moreover, Vit D deficiency may worsen osteopenia, causing rickets or its adult equivalent, osteomalacia. This disorder predominantly involves long bones and the axial skeleton (Bennett, 1976; Johnson and Watson, 2000). In the present patient, lameness, inability to do any activity and the reduced bone density of the scapula and bone structures surrounding the thorax were also observed. Therefore, the present patient was not only suffering from NSHP and rubber jaw, but also from osteomalacia due to Vit D deficiency.

The widespread use of commercial pet food has decreased the prevalence of nutritional bone disease over the last decades. More recently, however, veterinarians and pet owners have become more interested in the use of homemade and raw food diets. The pet owner's motivations for providing these non-commercial diets have included a desire to pamper the pet, control over the ingredients, avoidance of artificial preservatives, preservation of natural enzymes and phytonutrients, and efforts to achieve medical benefits (Laflamme *et al.*, 2008). However, the use of recipes not designed for pets, failure to follow the recipe, and deviation from the recipe over time are major areas of concern with homemade diets because they often lead to malnutrition in pets (Remillard, 2008). Furthermore, less than a third of the owners that feed their pet a homemade diet use a recipe designed for pets (Rahman and Yathiraj, 2000; Laflamme *et al.*, 2008), and this increases the risk for nutritional imbalances and nutritional bone disease. In the present study, no recipe designed for pets had been used by the owner pre-treatment. Post-treatment, the dog was recovering well, as the dog gained weight and was able to chew. The jaws were still swollen but firm, and the teeth were immobile. PTH was also markedly decreased and remained within the reference range, whereas 25-OH Vit D was clearly raised, thought it still did not reach reference values. This was due to a lack of owner compliance, as the owner changed the patient's diet over time by eliminating the vitamin and mineral premix from the diet. This once again resulted in Ca, P and Vit D intake below the NRC recommendations (2009b), yet the owner could not be convinced 'even' at this point to follow the earlier feeding advice.

This case report clearly shows the importance of obtaining pet owner commitment. Everyone who feeds the pet has to recognize, accept and understand the reason why the dietary treatment is necessary and commit to accomplishing the proposed goal. If not, it is impossible to achieve successful dietary management. According to the American Animal Hospital Association (AAHA, 2003), compliance with feeding therapeutic foods was only 19% in dogs for the treatment of six canine conditions (kidney disease, bladder stones or crystals, food allergies, chronic as well as acute gastro-intestinal disease, and obesity) and 18% in cats for the treatment of seven feline conditions (the same six canine conditions plus feline lower urinary tract disease). More than 11.6 million dogs and nine million cats with one of the diagnosed conditions were not fed an appropriate therapeutic diet at all or were not fed the diet for an appropriate period of time. When all pets with diagnoses that could benefit from dietary treatment were considered, overall compliance was only 5 to 7% (AAHA, 2003). In addition, it is also disturbing that 55% of pet owners who actually fed the therapeutic food also added other foods or treats to the recommended diet. The primary reason cited by clients was that they did not know this was not permitted. According to this AAHA Compliance Study, cost was not a major barrier to adherence, as only 4% of the pet owners either discontinued or refused therapeutic foods because of their cost. However, pet owners did cite insufficient client communication and education as the most important reason for noncompliance (AAHA, 2003). Nonetheless, even with appropriate veterinarian-client communication, the owner's motivation determines the success of any dietary treatment.

CONCLUSION

In conclusion, despite the widespread use of commercially formulated pet foods, owners sometimes still prefer non-commercial diets, often without taking into account the specific dietary requirements of their pet, thus increasing the risks for health problems such as nutritional bone diseases. NSHP and rickets/osteomalacia occur only rarely in dogs and cats, but they still have to be included in the differential diagnosis of metabolic bone disease in both young and adult pets, especially

in cases of unbalanced homemade diets. Furthermore, the effectiveness of any dietary treatment critically depends on the willingness of the owner to 'buy into' the plan and, in the case of homemade diets, not to deviate from the recipe.

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