Nutritional Approach to an unbalanced diet and subsequent overcorrection in a female kitten

Introduction

Growing cats have distinct nutritional requirements due to the fact that growth requires a specific amount of nutrients. In addition, the sensitivity to nutritional imbalances is high (Gross et al. 2010). Feline nutritional secondary hyperparathyroidism (NSH) is a metabolic disorder, which mainly occurs in young cats, but occasionally affects adults (Pedram et al. 2014). It was correlated with an increased calcium (Ca) demand necessary for bone growth (Hazewinkel 2012). NSH is caused mainly by diets low in Ca, high in phosphorus (P) and low in vitamin D or a combination of all, leading to an inverse Ca:P ratio (e.g. 1:16) (Lenox et al. 2015, Corbee 2017, Bing 2017, Langley 2012). Low ionized Ca (iCa) plasma concentrations, reflecting inadequate dietary Ca uptake, will induce continuous secretion of the parathyroid hormone (PTH). High PTH concentrations in the blood circulation will induce 1) increasing efficacy of active intestinal Ca absorption mediated by the 1,25-dihydroxyvitamin D release, 2) decreasing renal Ca excretion, 3.) inhibiting P reabsorption and 4) mobilizing Ca from bones by osteoclastic activation (Erben 2005, Parker et al. 2015, Corbee 2017). Consequently, chronic bone Ca resorption develops, leading to fibrous osteodystrophy. In dogs, most affected patients' exhibit symptoms related to osteopenia (due to chronic Ca deficiency), pathological fractures, lameness, stiffness, and reluctance to move. (Jamisidi et al. 2014, Langley 2012). Additionally, in dogs and cats, reported radiographic findings include bone deformation of scapulae and spine, spontaneous greenstick fractures of long bones, compression fractures in cancellous bones, and deformations of flat bones (Tomsa et. al. 1999, Hazewinkel, 1989, Nap & Hazewinkel, 1994, Tal et al. 2018). In some cases, kittens show symptoms related to acute hypocalcemia such as muscle tremors and seizures (Tomsa et al. 1999). Differential diagnoses for NSH include renal secondary hyperparathyroidism, osteogenesis imperfecta and rickets (absolute vit. D deficiency and low or normal Ca levels) (Parker & Chew 2015, Bing 2017). The therapeutic approach is based on a nutritional correction by feeding a complete and balanced diet providing sufficient energy density, protein, taurine, fat and essential fatty acids, linoleic acid (LA), α-linolenic acid (ALA), arachidonic acid (AA), docosahexaenoic acid (DHA), Ca, P, vit. D, potassium, sodium, zinc, copper and magnesium contents according to the requirements for growing kittens (Gross et al. 2010). Additional Ca supplementation (50 mg Ca/kg BW) for 3 weeks, is currently recommended for dogs (Hazewinkel 2012). In adult cats, such high supplementation could result in hypercalcemia, leading to kidney injury, impaired renal function and soft tissue calcification (Stockman et al. 2021). Vit. D

supplementation should be approached cautiously and only in cats with severe hypocalcemia. Excessive vit. D supplementation can cause hypercalcemia. The recommended dose of vit. D3 in cats is 0.03 to 0.06 µg/kg (Zoran 2002). Movement restriction through cage rest is indicated. After mineralization of the bones is completed, corrective surgery can be considered (Hazewinkel 2012, Parker et al. 2015, Corbee 2017, Zambarbieri et al. 2023). Up to 86 % of the frequently prescribed homemade recipes for cats show nutritional inadequacies in the mineral and vitamin composition for all respective life stages. Some of these nutritional inadequacies on homemade recipes include crude protein, choline, iron, thiamine, zinc, manganese, vit. E, copper, Ca and vit. D deficiency and often, vit. A excess, due to the inclusion of liver in the diets (Wilson et al. 2019, Villaverde & Chandler 2022). The long-term effects of vit. A dietary excess in cats are characterized by formation of extensive bony osteophytes and exostoses around joints, at the site of the tendon, the ligament and the joint capsule attachments causing intermittent lameness and ankylosis of the cervical and thoracic spine. (Polizopoulou et al. 2005, Kamphues et al.2014). Inadequate Ca supply, unsuitable Ca:P ratio and both shortage as well as excess of vitamin A and D quickly manifest and may potentially develop into disease such as NSH (McMillan et al. 2006, Tomsa et al. 1999, Wilson et al. 2019, Villaverde & Chandler 2022). Discrepancies and a lacking on consistent information specific for growing cats with NSH regarding nutritional recommendations, diagnostic tools, and blood normal ranges show the importance of this case report, illustrating the nutritional approach to a growing cat with initially misdiagnosed NSH. Also, the often-seen dietary over supplementation of vit. D and vit. A in homemade diets of kittens and cats leading to permanent skeletal deformities and impaired growth.

Clinical history

An intact European shorthair kitten (8 months old at the time of presentation) of Romanian origin was referred by the internal medicine clinic, to the Institute of Animal Nutrition and Dietetics (both of the Vetsuisse Faculty, University of Zurich) (Table 1, D -8). It was presented with a history of pathological fractures, marked skeletal deformations and neurological symptoms. Besides these signs, it appeared to be in a good body condition (weight at presentation 2.2 kg, body condition score 5/9 according to Laflamme 1997 for adult cats). No muscle loss was reported. Considering that during normal healthy development, lean female cats reach about 85% or more of their expected adult weight by eight months of age (Kamphues et al. 2014, Gross et al. 2010, Opsomer et al. 2022, Häring et al. 2011), the expected adult weight for this kitten was estimated at 2.5 kg, taking into account an impaired growth due to

nutritional imbalances (see nutritional approach). The results of subsequent blood results are shown in Table 1. Following the first confirmed pathological fracture at 4 months of age (D -203), PTH levels without further blood work-up were measured. Based on the results, the private veterinarian prescribed vit. D3 supplementation at an unknown initial dosage. This was increased to 400 IU/day following a subsequent fracture at 5 months of age (D -116). More rigorous diagnostic testing showed elevated alkaline phosphatase (ALP), and low 25-hydroxyvitamin D levels. The parameters of renal functionality within normal ranges allowed to rule out a secondary renal hyperparathyroidism. (Table 1, D -116). Two months later (Day -51) at 7 months of age, an additional check-up was performed. Added to the previous findings, a decreased vit. A level was found. Supplementation was advised in response to low serum vit. A and 25-hydroxyvitamin D concentrations (see nutritional approach). Due to continuous abnormal gait and lameness, the owner sought advice with the internal medicine clinic of the Vetsuisse Faculty, University of Zurich (D -8). The noted axis deviation of the right tibia and left femur confirmed the initial observations made by the private veterinarian. The x-rays revealed generalized osteopenia, malformation of multiple bones, and malunion healed folding pathological femoral fracture within the distal metaphysis. Only Ca, P and vit. B12 blood parameters were initially examined and found within normal ranges. A week later (Day 0) complementary blood tests revealed increased pancreatic lipase, PTH, and 1,25-dihydroxyvitamin D concentrations, however the 25-hydroxivitamin D concentrations were within the reference ranges internally set by the laboratory (Table 1, D 0). An additional abdominal ultrasound revealed a low-grade pancreatomegaly of no clinical relevance. Other presumptive diagnoses, such as rickets, where usually hypocalcemia and hypophosphatemia but pathological fractures are rather unusual (Evason et al. 2007) and osteogenesis imperfecta, where serum Ca and PTH levels are expected to be normal and there is typically medullary sclerosis and generalized muscle atrophy, were considered less likely. Based on the nutritional and clinical history and the above-mentioned diagnostic work-up, the diagnosis of NSH was established. No pharmacological treatment was prescribed and the patient was then referred to the Institute of Animal Nutrition and Dietetics for a nutritional consultation.

Nutritional approach

The nutritional contents of the patient's subsequent diets are specified in Table 2 and were calculated with Diet Check Munich ©2005 Version 3.0 (RV Software; based on NRC 2006, modified by Dobenecker and Kienzle). Diet A (diet fed before referral to the Institute of Animal Nutrition and Dietetics from D -51 on. Data on previously fed diets was not available) consisted

of a homemade diet based on three protein sources, different carbohydrate sources, assorted vegetables, and avocados occasionally provided. The diet was supplemented daily with a mineral vitamin powder, mineral vitamin tablets, and vitamin D_3 drops (see Table 3, Diet A). Important imbalances were found in Diet A, including a suboptimal Ca:P ratio of 2:1 with a vit.A, D and E over-supplementation and a low protein, energy density and essential fatty acids supply (Table 2, Diet A). Considering a delayed growth given the NSH diagnosis (Zambarbieri et al. 2023), the patient's adult ideal weight of 2.5 kg was calculated based on a growth curve for an average lean female cat, with a current BCS of 5/9 and 2.2 kg current body weight. Due to both the owner's and the patient's preferences, the homemade diet was only adapted. The adaptations on diet B (Table 2) addressed corrected for these imbalances. Avocados were eliminated from the diet due to their toxic potential in different species, including cats (Kovalkovičová et al 2009, Buoro et al 1994). To prevent excessive compensatory growth, the energy content and density of the diet were only slightly increased (±10%) under reservation of necessary adjustments depending on the further progress and weight development. The prescribed energy content corresponded to 80 % of the energy requirement for healthy kittens up to 9 months according to the FEDIAF (2021) and NRC (2006) guidelines [1.75 or 2 × (418 kJ ME × BW $kg^{0.67}$)]. The protein content was increased, in order to cover the crude protein (nitrogen) and amino acids requirements. During growth, especially on NSH cases, sufficient protein, thus protein-calorie malnutrition should be avoided to promote lean muscle tissue development and growth (Gross et al. 2010). Additionally, kittens show better growth rates when fed only high-quality protein, i.e., protein with an ideally matching amino acid pattern (Iben et al. 2021, Fascetti & Delaney 2012, Morris 2002). As a result, only high-quality (highly digestible of good biological value) protein (like chicken meat and codfish) was included in diet B (Table 3). Due to the low-degree pancreatomegaly, the fat content of the diet was reduced, should a pancreatic pathology develop (Rutgers & Biourge, 2010). However, a sufficient supply of essential fatty acids was ensured. Initially, walnut oil was chosen as an essential fatty acids source. EPA and DHA were supplemented as algae oil, due to their role in growth and development, and their additional well-known anti-inflammatory effects (Lenox & Bauer 2013, Lenox 2016). Dosages of up to 600-700 mg/d EPA + DHA have been tested in cats without detrimental effects, and anti-inflammatory response. However, the long-term safety of omega-3 fatty acid supplementation has not yet been determined in cats (Bauer 2011), and more conservative dosages (30-50 mg/kg) are usually recommended (Lenox 2016). The algae oil, provided 209 mg and 369 mg of EPA and DHA respectively. The omega-6 to omega-3 polyunsaturated fatty acid ratio of the diet was estimated to be 1.6:1, and the total amount of

AA was estimated at 4.65 mg. Dicalcium phosphate, meat bone meal, and a vitamin-mineral supplement were added to the diet B and replaced the supplements previously fed in diet A (Table 3). Ca and P supply was reduced to a tolerable range while further supporting bone mineralization, and the Ca:P ratio was with 1.1:1 decreased and, established within the recommended physiological range for growing cats of 1-1.5:1 (Gross et al. 2010) to avoid a relative Ca deficiency (Iben et al. 2021). Furthermore, the vit. A, D, and E supply was drastically reduced to achieve a maximum supplementation of threefold of the calculated recommended allowance (CRA) for the patient (Table 2). Since the patient liked vegetables, zucchini was kept in diet B (Table 3). The fiber sources should potentially favor the patient's gastrointestinal health (Sunvold et al. 1995, Wong & Gibson 2003). It was advised to feed the daily diet well mixed, as multiple small meals (2-3) (Fascetti & Delaney, 2012)

Follow-up

Two (D 13) and 6 weeks (D 48) after initiation of diet B, the owner reported clear and continuous improvement of the patient's demeanor and physical activity (ability to walk and jump). Two months after the initiation of diet B, (Table 1, D 75), the clinical improvement was confirmed by normalization 1,25-dihydroxyvitamin D and PTH- values (although not all within reference values), as well as improved bone mineralization on x-ray imaging. With the improvement due to nutritional management, the diagnosis of NSH could be confirmed. Radiographic examinations showed a slight increase in bone density and cortical bone thickness, metaphyseal ends of the long bones continued to widen, but the physeal plates appeared thinner. Hind limb and vertebral deformities persisted. Additionally, mild osteopenia in regression when compared to the initial x-rays performed on D 0 was found. The patient however had not gained weight and previously noted deformities had scarcely improved.

Three months after the initiation of diet B (D 103), at the age of 1 year, the diet was adjusted to meet the recommendations of an adult cat with an ideal weight of 2.5 kg (Table 2, Diet C). About 2 weeks after the initiation of diet C (D 116), the patient presented a recurrence of hind limbs and lumbar pain. The diet had to be adapted again to diet D1 and D2 .At owner's request, and to improve the diet palatability, the previous fat source was replaced by either fat richer meat (Table 3, Diet D1) or additional animal fat (Table 3, Diet D2), increasing the total fat content of the diet. The Ca and P supply was adapted to the requirements of a growing cat, the amount of vit. D was maintained and the protein content was reduced when compared to diet C (Table 2). Two months after the adaptation to diet D (D 202), at 1.2 years of age, only an elevation in the 25-hydroxyvtamin D concentration was found (Table 1, D 202). Three months

after the adaptation to diet D (D 236), 1,25-dihydroxyvitamin D levels were increased and PTH levels decreased in contrast to the last control lying within the laboratory reference values. Seven months after the adaptation to diet D (Table 1, D 357) at 1.8 years of age, an ovariectomy was performed. Pancreatic lipase was slightly over, whereas creatinine and Ca were slightly below the reference values. Results for 25-hydroxyvitamin D and 1,25-dihiydroxyvitamin D levels are pending. As osteopenia had markedly improved but is still present, adjustment of Diet D to adult requirements is not yet recommended.

Discussion

As early diagnosis and prompt response are crucial to a full recovery (Parker et al 2015) from NSH. It is regrettable that the necessary work-up to diagnose NSH, comprising a detailed nutritional history and blood examination including, urea, creatinine, total Ca, iCa, inorganic P, PTH, 25-hydroxyvitamin D, 1,25-dihydroxyvitamin D and radiologic diagnostic imaging (Zambarbieri et al. 2023) was only initiated at 8 months of age. The initial diagnosis relied only on the PTH concentrations below the reference range provided by the laboratory (Table 1, D -203). In both NSH and vit. D deficient rickets, PTH concentrations are often markedly elevated in an attempt to maintain iCa concentrations. Methods for the determination of feline PTH concentrations are limited, not widely available and relatively unreliable (Zambarbieri et al. 2021, Pineda et al. 2012, Smit et al. 2019). Normal cats often have PTH concentrations below the level of detection of currently used assays (Parker et al. 2015). In this case, an inaccuracy in the initial PTH determination is possible, (Table 1, D -203) and might have led to an inconclusive initial diagnosis. A thoroughly conducted diagnostic pathway to discard other presumptive diagnoses such as secondary renal hyperparathyroidism, osteogenesis imperfecta, acquired or congenital vitamin D disorders (rickets), hypovitaminosis and hypervitaminosis A is consequently essential when metabolic bone diseases are suspected (Bing 2017, Corbee 2017). The highly unbalanced diet fed during the first 5 months of life, based on an online recipe was most likely poor in Ca and rich in P (all-meat diet), resulting in an inappropriate Ca:P ratio. In this case, the initial Ca deficiency led to the development of NSH, which added to a dietary deficiency of vit. D could have contributed to the initial decrease of 25-hydroxyvitamin D (Zambarbieri et al. 2023). The chosen nutritional approach was a dietary adaptation to the recommended allowances of a growing cat (Table 2, Diet B). At 8 months of age, most cats are considered fully grown, however it has been observed that further weight stabilization and BCS still occurs later (Opsomer et al. 2022), therefore the energy density of diet B was only slightly increased when compared to diet A (Table 2). There was no weight

gain reported, possibly due to the retarded growth caused by NSH. The energy content of diet B could have been increased to a greater extent, however, given the fact that the patient was always in an ideal body condition, a further energy density increase was considered unnecessary. In diets C and D (Table 2) an increase in energy density was not incorporated since the patient was able to maintain an ideal body condition and ideal BCS. Diet B provided protein surplus, to support bone and further muscle growth. On diets C and D, the protein content was reduced but still covered the CRA for growth. It is possible, that the reduction in total protein, together with the decrease in the amount of Ca and P in diet C (Table 2 and 3) may have played a role on the recurrence of symptoms (D 116). Considering that cats have a high endogenous glucose demand met by obligatory amino-acid based gluconeogenesis (Rogers et al. 1977, Eisert 2011) that might increase under the influence of stressors, such as trauma, toxins or infection (Laflamme & Hannah 2012), in this case NSH. Diets B, C and D provided sufficient essential fatty acids. Additional to ALA and LA, due to the low Δ6 desaturase activity, AA is considered essential in cats, especially during reproduction and growth (NRC 2006, Bauer 2006, Villaverde & Fascetti, 2014, Pawlosky et al. 1997). Although there is no clear evidence that EPA and DHA are required by cats, these have a potentially beneficial role in osteo-articular diseases and inflammation (Hazewinkel 2012). The total fat content on diet D (Table 2) was increased when compared to diet B to improve the palatability of the diet. Cats tolerate relatively high amounts of total fat in the diet (Villaverde & Fascetti, 2014). However, it is important to consider the echographic findings associated with a pancreatopathy. In the latest blood tests (Table 1, D 357) a slight elevation of pancreatic lipase was observed, suggesting that a future reduction in the total amount of fat in the diet might be appropriate in this case (Rutgers & Biourge 2010). Diet A (Table 2) had numerous nutritional imbalances, including Ca, P, vit. A, D and E excess. The amount of Ca included in the diet was about three times the CRA and the amount of P about double the CRA. For vit. A, the dietary excess was over 17 times the CRA. The calculated vit. A safe upper limit (SUL) for this patient is about 5 times that amount (23311 IU) or about 97 times the CRA. Reported effects of vitamin A excess in growing cats include shorter and osteoporotic long bones and damaged epiphyseal plates. Additionally, teratogenic effects in pregnant queens have been reported. (Clark 2021, Freytag, 2001). Adult cats consuming vitamin A in doses equivalent to the SUL do not show abnormal locomotion or clinical signs of liver failure after 18 months of supplementation. However, show subtle skeletal changes and liver pathology, suggesting that the current SUL for vitamin A for cats might be too high and requires revision (Corbee et al. 2014). For this case, the excessive vit. A could have perpetuated the bone deformities and worsened the

prognosis. In the subsequent diets (B, C and D) a vit. A supply close to twice the CRA was maintained for the patient. The measurement of vit. A blood values could provide additional information on vit. A playing a role in the radiological changes observed and the case's outcome. In this patient, acquired hypovitaminosis D (rickets) was initially hypothesized (Table 1, D-116) and therefore only a vit. D supplementation on top of a Ca deficient diet was initiated. Diet A posed a vit. D over supplementation of about 40 times the patients' CRA, far below the calculated SUL (2636 IU), about 3.5 times that amount or 146 times the CRA (Table 2, Diet A). Prior to nutritional adaptation, a decreased 25-hydroxyvitamin D value was evidenced (Table 2, D -116 and D -51) in spite of the vit. D over supplementation from diet A. To assess vit. D status in both human and veterinary medicine, 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D are primarily measured. However, the quantification of vit. D metabolites is not straightforward (Clarke et al. 2021). In a review on vitamin D it was stated, that 25-hydroxyvitamin D is not a very sensitive indicator of vit. D status in dogs and cats, due to the difficulty to extrapolate data from human medicine and measurement methodology differences. 25-hydroxyvitamin D measurement provides a rough estimation of dietary intake, while 1,25-dihydroxyvitamin D provides more insight in metabolism and effects on target issues (Corbee, 2020), if all mechanisms are reacting as they are in other mammals. Additionally, the reference ranges for different vit. D metabolites in cats have not been established. Serum 25-hydroxyvitamin concentration in cats, displayed ranges in control groups between 14.9 to 83.1 ng/ml, and in diseased cats, between 1.7 to 97.1 ng/ml (Zafalon et al. 2020). These may also be due to the different methods used. Age-related differences have been reported as well, where 25-hydroxyvitamin concentrations in kittens between 3 and 6 months old, are significantly higher than concentrations of older kittens and adult cats (Parker et al. 2017) and that the 1,25-dihydroxyvitamin D values in growing kittens are higher than previously thought (Pineda et al. 2013). Currently, reliable age-related reference intervals for the vit. D metabolites are required in kittens, to support an accurate diagnosis in cases as the one here presented. Vit. D supplementation in diets B, C and D was maintained at about three times the calculated CRA (Table 2). However, the patient continued to exhibit elevated 25-hydroxyvitamin and 1,25-dihydroxyvitamin D levels over time (Table 1). These elevated serum vit. D concentrations may be related to an incomplete bone mineralization process. On the other hand, plasma 25-hydroxyvitamin concentrations tend to increase with abnormally high vit. D content in the diet (Morris et al. 1998), meaning that the vit. D content on diet D may be too high for this patient, or that as previously discussed, more precise and accurate methods for the vit. D status determination and reliable reference intervals for cats are indispensable. The reported

mineralization time for NSH patients after nutritional correction is 6 to 8 weeks (Parker et al. 2015). However, for this patient the bone mineralization process is still ongoing. The Ca and P supply was maintained at about 2.7 times the CRA respectively for an adult cat and an optimal Ca:P ratio of 1:1. Additionally, more specific and sensitive methods for the accurate quantification of bone mineral density in cats when compared to x-rays, such as dual-energy x-ray absorptiometry (DEXA) and computed tomography (CT) are available (Dimopoulou et al 2010), and could be of much greater diagnostic importance to determine the bone mineralization process.

Conclusion

A large proportion of the homemade diets recommended for cats on the internet and books used by veterinarians are unbalanced and may result in serious developmental diseases. When suspecting NSH, a thorough nutritional history is required in addition to a comprehensive clinical assessment, to exclude presumptive diagnoses and therefore false or incomplete treatment. Early recognition through a diet check and adequate nutritional management are main factors to improve the patient's prognosis. Further work integrating blood concentrations of vit. D metabolites is required, especially considering the great variability between methods and the absence of age-appropriate reference ranges. When early diagnosed, NSHs' effects may be reversed allowing a good prognosis. Following nutritional correction, sensitive and specific methods such as DEXA and pQCT are valuable to assess the bone mineralization process and should be considered if possible.

Tables

Table 1: Blood results before and after referral to the internal medicine department of the Vetsuisse Faculty, University of Zurich. Measurements using differing reference values are given per level. The day of initiation of the diet as recommended by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zurich is taken as day (D) 0.

	D -203	D -116	D -51	D -8	D 0	D 75	D 202	D 236	D 357	Reference
Amylase			1233							< 1850 U/l
Lipase (DGGR)			23.4		23				26	6- 21 U/l
Fructosamine			344							< 340 µmol/l
Triglycerides			0.52						0.5	< 1.14 nmol/l
Cholesterol			4.7						3.4	1.8-3.9 mmol/l
T-Bilirrubin			0.8						<2.5	< 3.4 µmol/l
Alkaline phosphatase		275	192						16	16-43 U/l
g-GT		5	< 0.1				4			<5 U/l
GPT/ ALT		19	27.4				37		38	34-98 U/l
GOT/AST		10	12.6				14		26	0-36 U/l
CK			68						224	77-355 U/l
T Protein		6.1					6.5			5.2-8.8 g/dl
			64.9						65	57-94 g/l
Albumin			42.1			39			39	26-56 g/l
Globulin			22.8						26	< 55 g/l
Urea		18					31			14-38 mg/dl
			6.9		8	6.3			12.6	6.5-12.6 mmol/l
Creatinine		0.8					1.1			0.4-2.3 mg/dl
			83		85	98			61	98-163 µmol/l
Phosphate		2.1	2.1	1.91		1.39		1.41	1.48	2.45-2.55 mmol/l (kittens, Pineda et al. 2013) 0.9-1.8 mmol/l (adults)
							4.3			1.6-8.1 mg/dl
Magnesium			1			0.90				0.6-1.3 mmol/l
							2.4			1.6-2.7 mg/dl
Calcium		2.4	2.3	2.50		2.49	2.50	2.63	2.38	2.3-3.0 mmol/l
Ionized Calcium		1.18								1.4-1.8 mmol/l
Sodium			157			150	150		152	145-158 mmol/l
Potassium			4.9			3.19	3.9		3.9	3.0-4.8 mmol/l
Iron			44.5							8-31 µmol/l
Vitamin A (HLC)			179							180-250 µg/l
25-hydroxyvitamin D		39.67	39.9		144		206.63			126-163 nmol/l (50.4-65.2 ng/ml)
1,25-dihydroxyvitamin D					891	673		449		52-104 pmol/l (21.6-43.3 pg/ml)
Vitamin E (HLC)			17							> 3.5 mg/l
Vitamin B12 intern	650			620						225-1452 pmol/l
T4 total			3.77				1.95			1-4 µg/dl
						29.4				16-46 nmol/l
PTH	2.88				59	43		21		0-20 pg/mL
Canine TSH						0.27				ng/ml

Table 2: Comparison of subsequent diets nutritional content, calculated with Diet Check Munich © 2005 Daily values of Nutrient content of the diets provided. CRA = Calculated Recommended allowance; Diet A = homemade diet fed before the dietary consultation from D -51 on; Diet B = completely balanced homemade diet for growing cats; Diet C= homemade diet adapted for adult requirements of the patient. Diets D1 and D2: homemade diets adapted to support further bone mineralization. Diets B, C, D1 and D2 prescribed by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zurich. The day of implementation of the diet is given between brackets. The day of initiation of the diet as recommended by the Institute of Animal Nutrition and Dietetics of the Vetsuisse Faculty, University of Zurich is taken as day (D) 0

	CRA Growth	Diet A (D -51)	Diet B (D 0)	CRA Adult	Diet C (D 103)	Diet D1 (D 116)	Diet D2 (D 116)
Energy (MJ ME)	0.9	0.8	0.9	0.7	0.7	0.7	0.8
Energy density (MJ/100 g DM)	/	1.2	1.7	/	1.9	2.0	2.2
Crude Protein (g)	23	23	35	13	25	19	25
Nitrogen free extract (g)	/	33	17	/	5.6	0	2.7
Fat (g)	/	10	4	/	7	11	7
Fat (% Dry Matter)	/	15.2	7.5	/	19.4	31.4	18
Methionine (g)	0.40	0.51	0.82	0.08	0.59	0.51	0.59
Cystine (g)	0.40	0.28	0.37	0.08	0.27	0.24	0.27
Taurine (g)	0.036	0.1	0.1	0.1	0.2	0.1	0.1
Calcium (mg)	548	1621	597	133	254	486	490
Phosphorus (mg)	493	799	536	118	175	408	415
Magnesium (mg)	27	65	80	19	52	38	49
Potassium (mg)	274	593	503	241	400	325	360
Sodium (mg)	96	91	94	31	73	71	69
Iron (mg)	5.5	10.9	6.9	3.7	7.1	4.0	4.6
Copper (mg)	0.6	1.5	1.2	0.2	0.6	0.8	0.8
Zinc (mg)	5.1	16.4	6.1	3.4	8.8	4.0	4.0
Chloride (mg)	61	137	166	44	165	105	123
Iodine (µg)	151	468	139	65	139	90	95
Vitamin A (IU)	243	4285	563	154	269	465	379
Vitamin D (IU)	18	720	53	13	50	36	36
Vitamin E (mg)	3	56	5	2	4	3	3
Vitamin B1 (mg)	0.07	3.73	0.23	0.26	0.98	0.19	0.16
Vitamin B12 (µg)	2	15	6	3	5	1	14

Table *3*: Quantity of ingredients in Diet A (diet fed previous to the nutritional intervention). Diet B (as recommended during growth by the Institute of Animal Nutrition of the Vetsuisse Faculty, University of Zurich). Diet C (as recommended for adult requirements by the Institute of Animal Nutrition of the Vetsuisse Faculty, University of Zurich), and Diet D1 and D2 (as recommended for adult requirements with an ongoing bone mineralization process by the Institute of Animal Nutrition of the Vetsuisse Faculty, University of Zurich)

	Diet A	**Diet B**	**Diet C**	**Diet D1**	**Diet D2**
Chicken meat 10 % Fat (raw weight) (g)	77	/	/	85.7	/
Chicken breast 1 % Fat (raw weight) (g)	/	129	94.6	/	95
Cod fish 1 % Fat (raw weight) (g)	13	21	15.4	14.3	15
Pork lard (g)	/	/	/	/	4
Egg (raw weight) (g)	7	/	/	/	/
Whole wheat pasta (raw weight) (g)	10	/	/	/	/
Rice (raw weight) (g)	10	/	/	/	/
Boiled potatoes (cooked weight) (g)	10	/	/	/	/
Zucchini (raw weight) (g)	20	20	20	20	20
Pumpkin (raw weight) (g)	10	/	/	/	/
Red bell pepper (raw weight) (g)	10	/	/	/	/
Mushrooms (raw weight) (g)	20	/	/	/	/
Rapeseed oil (g)	1	/	/	/	/
Walnut oil (g)	/	2	4	/	/
Algae oil (g)	/	1	2	2	2
Dicalcium phosphate (g)	/	1	0.5	1.0	1
Trace element supplement powder 1 (g)	10	/	/	/	/
Trace element supplement powder 2 (g)	/	1.5	1.0	1.0	1
Trace element supplement tablets (Tabs.)	1	/	/	/	/
Vitamin D supplement (400 IE) (drops)	4	/	/	/	/

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