Seizures and severe nutrient deficiencies in a puppy fed a homemade diet

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Case Description—An 8-month-old male Saint Bernard developed tetanic seizures and hyperthermia during evaluation of bilateral osteochondritis dissecans of the shoulder joints. Further investigation revealed that the dog was receiving an unbalanced homemade diet.

Clinical Findings—Preliminary evaluation of the dog revealed bilateral signs of pain and mild muscle wasting in the shoulder joint areas. Serum biochemical analysis revealed severe hypocalcemia, hyponatremia, hypochloremia, hyperphosphatemia, vitamin D deficiency, and taurine deficiency. Diffuse osteopenia was identified on radiographs of the mandible and long bones, confirming bone demineralization. Analysis of the homemade diet revealed that the dog's diet was severely deficient in a variety of nutrients.

Treatment and Outcome—The dog responded positively to treatment for hypocalcemia, hyperthermia, and seizures. The dog's diet was changed to a complete and balanced canine diet formulated for growth. Body weight and body condition were monitored, and dietary intake was adjusted to achieve optimal body condition during growth. After initial evaluation, serial monitoring of serum calcium and taurine concentrations revealed that values were within reference limits and the dog had no further clinical signs associated with dietary deficiency.

Clinical Relevance—Findings in this puppy highlight the risks associated with feeding an unbalanced homemade diet during growth and the importance of obtaining a thorough dietary history from all patients. For owners who elect to feed a homemade diet, it is critical to have the homemade diet carefully formulated by a veterinary nutritionist to avoid severe nutrient imbalances, especially in young, growing dogs. (*J Am Vet Med Assoc* 2012;241:477–483)

An 8-month-old sexually intact male Saint Bernard was evaluated at the surgical service of the Tufts Cummings School of Veterinary Medicine Foster Hospital for Small Animals because of bilateral osteochondritis dissecans of the shoulder joints following a 2-month history of bilateral forelimb lameness. The dog was acquired from a breeder at 11 weeks of age, at which time it had diarrhea and was eating a chicken-based adult dry dog food (3/4 cup, q 8 h).[a] The owners were unaware of whether the dog had diarrhea before they acquired it. The diarrhea continued for 7 days after arriving at the new owners' home, at which time the owners began feeding the dog a homemade cooked hamburger and rice diet (diet proportions consisted of 2 lb of ground hamburger and 1 1/2 cups of white rice, q 8 h). The dog was also dewormed during the first 7 days of living with the owners. The dog's diarrhea resolved within 3 days after changing the diet, and the owner interpreted this improvement to mean that the dog had a food allergy (presumably to chicken) and decided to continue the homemade diet. After feeding the homemade diet for 7 days, the owners chose to provide dietary supplementation with a commercial product for dogs.[b] In an effort to make the homemade diet more complete, some human foods and a small amount of the chicken-based adult dry dog food were added (**Table 1**). Whenever the owner attempted to increase the small amount of the chicken-based adult dry dog food incorporated into the homemade diet, the dog reportedly had recurrence of diarrhea.

Abbreviations

AAFCO	Association of American Feed Control Officials
BCS	Body condition score
iPTH	Intact parathyroid hormone
NRC	National Research Council

This diet was fed for 5 months prior to the initial evaluation at the authors' hospital. During this time, the dog was seen by the referring veterinarian for vaccinations and deworming and was assessed to be in good body condition. No further diarrhea occurred following initiation of the homemade diet and deworming. At 7 months of age, the dog was evaluated by the referring veterinarian because of bilateral forelimb lameness with signs of pain localized to the shoulder joint region. Findings on radiographs of the shoulder joints were consistent with bilateral osteochondritis dissecans of the shoulder joints. Subchondral bone flattening of the caudal portion of the humeral head was evident in both shoulder joints. Osteopenia was not detected on these radiographs (**Figure 1**). The referring veterinarian prescribed oral administration of an NSAID as needed until the dog could be evaluated at the authors' hospital.

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Table 1—Diet composition of a homemade diet fed to a Saint Bernard puppy from 11 weeks of age until 8 months of age.

Ingredient	Amount consumed/d
Beef, ground, 90% lean meat/10% fat (pan cooked without added fat)	2 lb
White long-grain rice (boiled)	1.5 cups
Raw apple with skin	0.5 medium-sized apple
Broccoli (boiled without added fat)	0.33 cups
Chicken-based adult dry dog food[a]	0.33 cups
Whole raw egg (with shell)	0.43 egg (fed 3 times/wk)
Dietary supplement[b]	0.5 of manufacturer's scoop

Dry matter, 4,099.3 kcal/kg. Moisture, 64.4%. Proportions of metabolizable energy from protein, 38.78%; metabolizable energy from carbohydrates, 18.04%; and metabolizable energy from fat, 43.19%.

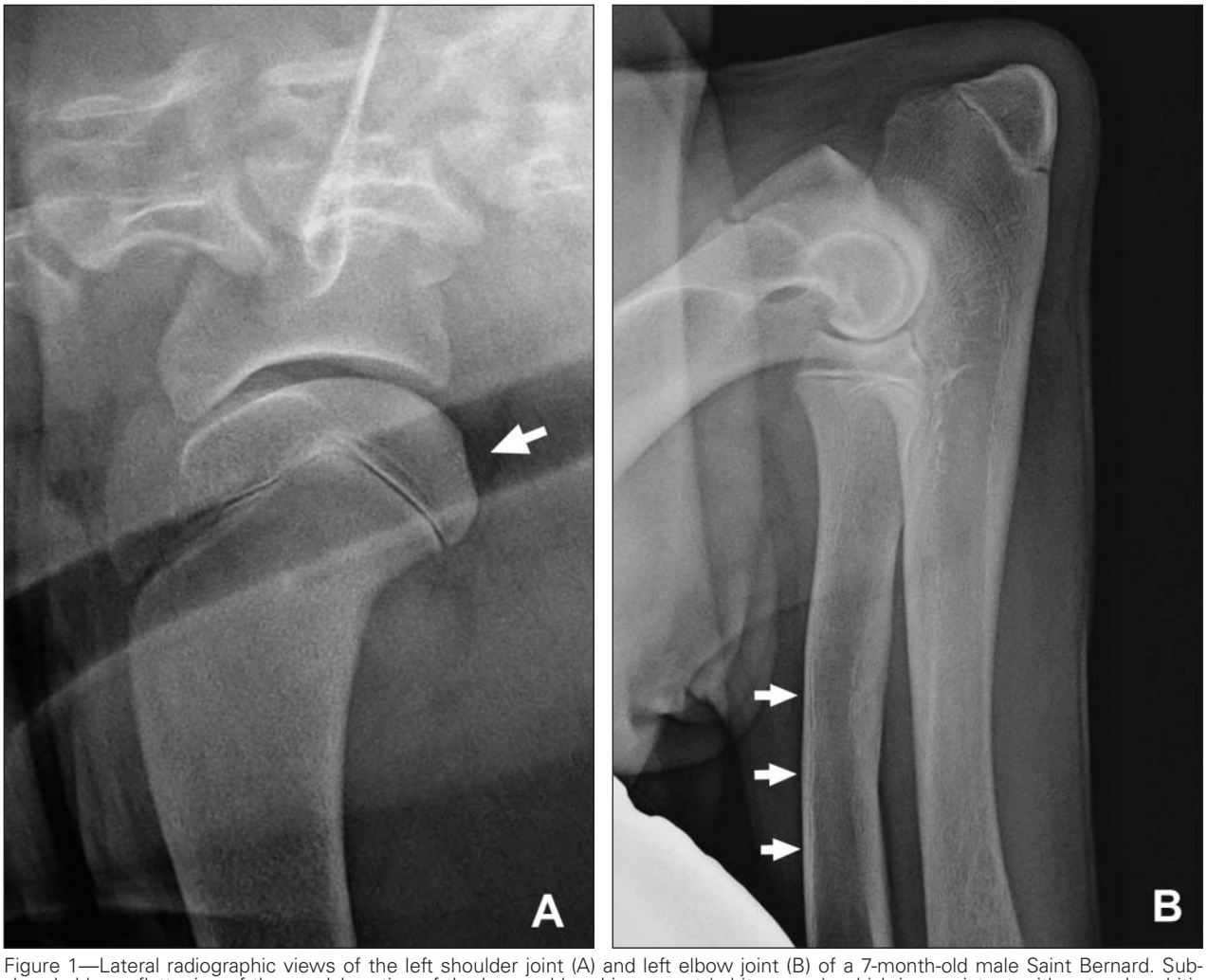

Figure 1—Lateral radiographic views of the left shoulder joint (A) and left elbow joint (B) of a 7-month-old male Saint Bernard. Subchondral bone flattening of the caudal portion of the humeral head is present (white arrow), which is consistent with osteochondritis dissecans of the shoulder joint. Although diffuse osteopenia was not detected, there is a mild diffuse reduction in cortical opacity and a double cortical line is present along the cranial border of the radius (white arrows).

On referral, the dog had bilateral forelimb lameness with signs of shoulder joint pain, a BCS of 4 of 9,[1] mild generalized muscle wasting, and a body weight of 44 kg (96.8 lb). No other abnormalities were noted on physical examination, but following the initial examination, the dog developed focal tremors that progressed to focal seizures. The dog became tachycardic (220 beats/min) and hyperthermic (39.4°C [103°F]) and was taken to the intensive care unit, where diazepam (0.5 mg/kg [0.23 mg/lb], IV, once), oxygen via a face mask, and lactated Ringer's solution (22.7 mL/kg [10.3 mL/lb], IV bolus, once) were administered.

Initial biochemical analysis revealed several electrolyte abnormalities, including respiratory alkalosis without compensation (venous pH, 7.5 [reference range, 7.33 to 7.47]; bicarbonate concentration, 24.5 mmol/L [reference range, 18 to 24 mmol/L]; and carbon dioxide concentration, 26 mEq/L [reference range,

14 to 28 mEq/L]), hypocalcemia (total calcium concentration, 7.7 mg/dL [reference range, 9.4 to 11.8 mg/dL]; ionized calcium concentration, 0.8 mmol/L [reference range, 1.2 to 1.4 mmol/L]), hyponatremia (137 mEq/L; reference range, 140 to 150 mEq/L), hypochloremia (90 mEq/L; reference range, 106 to 116 mEq/L), hyperphosphatemia (7.9 mg/dL; reference range, 2.6 to 7.2 mg/dL), hyperalbuminemia (4.4 g/dL; reference range, 2.8 to 4.0 g/dL), hypoglobulinemia (2.0 g/dL; reference range, 2.3 to 4.2 g/dL), high alkaline phosphatase activity (181 U/L; reference range, 12 to 127 U/L), and high creatine kinase activity (453 U/L; reference range, 22 to 422 U/L). Serum magnesium concentration was 1.8 mEq/L (reference range, 1.8 to 2.6 mEq/L), and potassium concentration was 3.8 mEq/L (reference range, 3.7 to 5.4 mEq/L). Findings on CBC were within reference limits. The dog's severe hypocalcemia was suspected to be the cause of the seizures, and a bolus of 10% calcium gluconate (45.5 mg/kg [20.7 mg/lb], IV) was administered over several minutes.

During the initial management of the seizures, the dog became cyanotic and its body temperature increased to 42.2°C (108°F). An additional bolus of calcium gluconate (68.2 mg/kg [31.0 mg/lb], IV) was administered over several minutes, and the dog was also treated with propofol (4.6 mg/kg [2.1 mg/lb], IV). Systolic arterial blood pressure ranged from 75 to 90 mm Hg. The dog was then intubated, and cold water gastric lavage was performed. Calcium gluconate (18 mg/kg/h [8.2 mg/lb/h], IV), diazepam (0.3 mg/kg/h [0.1 mg/lb/h], IV), and lactated Ringer's solution (2.5 mL/kg/h [1.1 mL/lb/h], IV) were also initiated as constant rate infusions. The dog remained intubated, and anesthesia was maintained with 3% isoflurane for an additional hour as body temperature decreased to 37.8°C (100°F). Systolic arterial blood pressure returned to 110 mm Hg after intubation. Inhalation anesthesia was then discontinued and the dog was extubated.

Point-of-care testing was used to measure clinicopathologic values at 2 hours and 6 hours after initial evaluation. By 6 hours after admission, acid-base and biochemical abnormalities seen at initial evaluation had been corrected except for hypocalcemia (ionized calcium concentration, 1.1 mmol/L; reference range, 1.2 to 1.4 mmol/L), hyponatremia (139.6 mmol/L; reference range, 147 to 154 mmol/L), hypochloremia (106 mmol/L; reference range, 109 to 120 mmol/L), and hypokalemia (3.3 mmol/L; reference range, 3.8 to 4.9 mmol/L). For the remainder of the dog's first day of hospitalization, administration of calcium gluconate, diazepam, and lactated Ringer solution was continued and the dog remained in the intensive care unit.

To determine the cause for the dog's severe hypocalcemia, which was thought to be due to either nutritional secondary hyperparathyroidism or primary hypoparathyroidism, blood was collected for analysis of iPTH, ionized calcium, and 25-hydroxyvitamin D concentrations. In addition, a detailed dietary history was collected from the owners and a computer analysis was performed to compare the nutrients in the homemade diet with dietary requirements for growth in dogs.[2,3] The diet had multiple and substantial deficiencies, including calcium, phosphorus, and vitamin D, all of which were < 50% below minimum requirements set by the NRC[3] as well as the AAFCO (**Table 2**).[2] Additional dietary deficiencies in sodium, chloride, iron, iodine, choline, copper, folic acid, vitamin A, and linoleic acid were identified. Although there are no established minimum dietary requirements for taurine in dogs, blood was also collected for analysis of serum and whole blood taurine concentrations because of the low amount of taurine in the homemade diet and previous studies[4–7] of dilated cardiomyopathy associated with taurine deficiency that have included Saint Bernards and other giant-breed dogs.

Intravenous administration of calcium gluconate and lactated Ringer's solution was continued until day 2 of hospitalization when the hypocalcemia resolved. Serum ionized calcium concentration was 1.3 mmol/L. Hyponatremia, hypochloremia, and hypokalemia also resolved by day 2. Serum sodium, chloride, and potassium concentrations were 142.6 mEq/L, 115 mmol/L, and 3.8 mmol/L, respectively. Serum ionized calcium concentrations were monitored to ensure serum calcium concentrations remained stable as the IV administration of calcium gluconate was gradually reduced.

Table 2—Comparison of the nutritional content of a homemade diet fed to an 8-month-old puppy to the NRC[3] and AAFCO[2] dietary recommendations for growth in dogs.

Nutrient/1,000 kcal	NRC minimum requirement*	AAFCO minimum requirement	Homemade diet
Protein (g)	35	62.9	96.94
Arginine (g)	1.33	1.77	6.25
Histidine (g)	0.5	0.63	2.87
Isoleucine (g)	1.0	1.29	4.13
Leucine (g)	1.63	2.06	7.21
Lysine (g)	1.4	2.20	7.43
Methionine-cysteine (g)	1.05	1.51	3.28
Phenylalanine-tyrosine (g)	2.0	2.54	6.52
Threonine (g)	1.25	1.66	3.5
Tryptophan (g)	0.35	0.57	0.42
Valine (g)	1.13	1.37	4.62
Crude fat (g)	21.3	22.9	47.99
Linoleic acid (g)	3.0	2.9	1.78
Calcium (g)	2.0	2.9	0.79
Phosphorus (g)	2.5	2.3	1.13
Potassium (g)	1.1	1.7	1.66
Sodium (mg)	550	860	370
Chloride (mg)	720	1,290	10
Magnesium (mg)	45	110	170
Iron (mg)	18	23	13.22
Copper (mg)	2.7	2.1	0.68
Manganese (mg)	1.4	1.4	0.75
Zinc (mg)	10	34	22.65
Iodine (µg)	220	430	80
Selenium (µg)	52.5	30	90
Vitamin A (retinol equivalents)	303	428.7	34.7
Vitamin D (µg)	2.75	3.57	0.02
Vitamin E (mg)	6.0	14	10.21
Thiamine (mg)	0.27	0.29	54.25
Riboflavin (mg)	1.05	0.63	3.19
Pantothenic acid (mg)	3.0	2.9	3.58
Niacin (mg)	3.4	3.3	23.98
Pyridoxine (mg)	0.3	0.29	2.35
Folic acid (µg)	54	50	0.17
Vitamin B12 (µg)	7	6	10
Choline (mg)	340	343	327.3
Taurine (g)	0.08†	0.25†	0.35

*Adequate intake reported for nutrients without published NRC minimum requirements. †No dietary requirement established for dogs; NRC and AAFCO allowances for feline extruded diets are provided for a point of reference.

Oral administration of calcium carbonate[c] (56.8 mg/kg [25.8 mg/lb], PO, q 6 h) was initiated as IV administration of calcium gluconate was being discontinued. While awaiting results of determination of iPTH, 25-hydroxyvitamin D, and taurine concentrations, administration of calcitriol[d] (5.7 ng/kg [2.6 ng/lb], PO, q 12 h) and taurine[e] (11.4 mg/kg [5.2 mg/lb], PO, q 12 h) was initiated on the second day of hospitalization. Also on day 2, feeding with a commercial diet[f] designed to meet the dietary requirements for growth in dogs was initiated to achieve estimated resting energy requirements (1,196 kcal/d divided into 4 meals [ie, approx two-thirds of a can {306 kcal}, q 6 h]) until the most appropriate long-term diet was established.[8] Although calorie requirements were likely higher because of the dog's age, a conservative calorie estimate was initially used during hospitalization to account for the physical inactivity.

To assist in determining the cause for the hypocalcemia, radiographs of the forelimbs that had been obtained by the referring veterinarian were reviewed. Although osteopenia was not apparent radiographically in the humerus or scapula, diffuse cortical thinning and a double cortical line were present along the cranial aspect of the radial cortex in the lateral view. This latter finding has been associated with osteopenia.[9] Because changes on the radiographs of the forelimb were subtle, radiography of the mandible was performed to further investigate the possibility of bone loss. The mandible is known to undergo demineralization in other situations of altered calcium homeostasis, such as renal hyperparathyroidism, which can result in osteomalacia marked by softening and degenerative changes of the jaw and facial skeleton (ie, rubber jaw).[10] Radiographic findings of the mandible were consistent with diffuse osteopenia (**Figure 2**). Radiographic features included decreased mandibular corticomedullary distinction, a partial loss of visualization of the lamina dura around the tooth roots, and only faint mineralization of the hyoid bones.

The dog was discharged from the hospital on day 3. Because of the owner's continued concerns that the dog had a food allergy to chicken, the owner was instructed to feed a fish-based commercial diet[g] that met the minimum requirements of the AAFCO for growth in dogs. Because the dog was in good body condition, the previous intake of homemade diet was used as a starting point for calorie requirements (2,080 kcal/d); therefore, an initial recommendation of 5.25 cups/d of the commercial diet was made (divided into 2 meals). The owner also was instructed to feed no added human or pet foods, supplements, or treats other than a controlled number of specific hydrolyzed dog biscuits.[h] In addition, only unflavored heartworm preventative and toothpaste were recommended because flavored heartworm and toothpaste can contain chicken, beef, or pork. While awaiting results of iPTH, 25-hydroxyvitamin D, and taurine testing, the administration of calcitriol (5.7 ng/kg, PO, q 12 h), taurine (11.4 mg/kg, PO, q 12 h), and calcium carbonate (56.8 mg/kg, PO, q 6 h) was to be continued.

Ionized calcium concentrations were reassessed 3 days after hospital discharge and were within reference limits (1.4 mmol/L; reference range, 1.2 to 1.4 mmol/L); therefore, calcium gluconate and calcitriol administration were discontinued. Results for determination of iPTH, 25-hydroxyvitamin D, and taurine were available at this time: iPTH concentration was within reference range (1.8 pmol/L; reference range, 0.5 to 5.0 pmol/L), but 25-hydroxyvitamin D concentration was low (18 nmol; reference range, 60 to 215 nmol), supporting a diagnosis of vitamin D deficiency and nutritional secondary hyperparathyroidism. In addition, whole blood (160 nmol/mL; reference range, 200 to 350 nmol/mL) and plasma (29 nmol/mL; reference range, 60 to 120 nmol/mL) taurine concentrations were low, supporting a diagnosis of taurine deficiency.

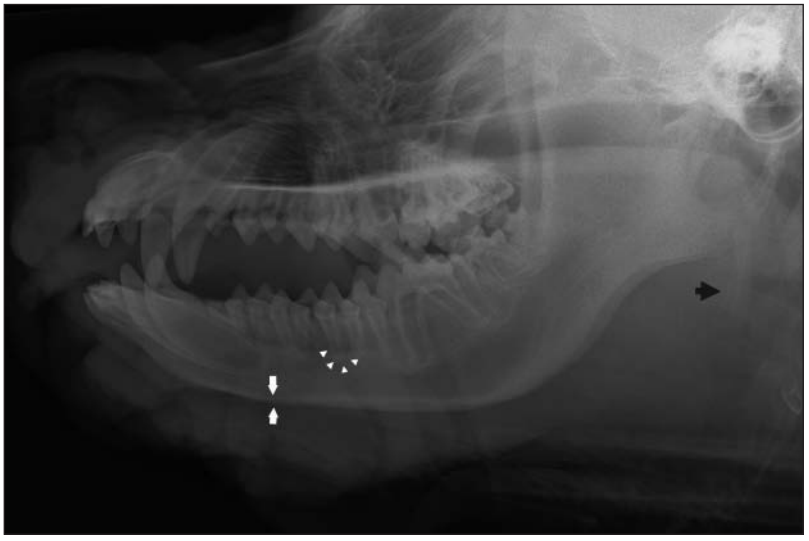

Figure 2—Right lateral radiographic view of the mandible and maxilla of the same puppy as in Figure 1; images were obtained 4 weeks later than those in Figure 1. There is decreased mandibular corticomedullary distinction (white arrows), a partial loss of visualization of the lamina dura around the tooth roots (white arrowheads), and only faint mineralization of the hyoid bones (black arrow). These changes are consistent with osteopenia.

An echocardiogram was performed 2 weeks after hospital discharge; contractile function and cardiac chamber size were within reference limits. Although taurine intake from the recommended fish-based commercial diet was 1,087 mg taurine/d, dietary supplementation with taurine (11.4 mg/kg, PO, q 12 h) was continued until serum taurine concentrations could be reassessed 4 weeks after hospital discharge.

The dog was weighed once weekly on the same scale and assessed for BCS, and the amount of food was adjusted to ensure a lean BCS (ie, 4/9) and optimal growth. All clinicopathologic values at 2 and 4 weeks following hospital discharge were within reference limits, including serum ionized calcium concentrations. Whole blood taurine concentration was reassessed 4 weeks after hospital discharge and had increased to 408 nmol/mL (reference range, 200 to 350 nmol/mL). As a result, dietary supplementation with taurine was discontinued. The dog's

initial mild bilateral forelimb lameness had resolved during the weeks following hospital discharge; therefore, no further diagnostic testing or treatment was pursued by the owner for this problem.

Discussion

The homemade diet fed to this growing giant-breed dog resulted in multiple and severe nutrient deficiencies. Of initial primary concern was the severe hypocalcemia resulting in seizures (with resultant hyperthermia and metabolic abnormalities). Calcium plays an important role in stabilizing sodium channels on axons of nerves and preventing muscle tetany. This role explains why the patient developed muscle tremors and eventual seizures because of severe hypocalcemia.[11] Although hypocalcemia was the most likely cause of the seizures, other causes of seizures were initially considered, such as idiopathic epilepsy, toxins, or head trauma. However, the severe hypocalcemia quickly narrowed the differential diagnoses to nutritional secondary hyperparathyroidism or primary hypoparathyroidism. In this case, a complete dietary history was key to aiding the clinicians because a homemade diet of this composition (ie, meat and rice) is known to have important nutrient deficiencies. The diagnosis of primary hypoparathyroidism was later excluded when an iPTH concentration within reference range was detected. Although the dog had been compensating for very low blood calcium concentrations for some time at home, a possible cause for acute seizures at our hospital could be respiratory alkalosis from panting, which decreased the dog's already low blood calcium concentration below the critical threshold, resulting in seizures.[12]

Many of the physical and clinicopathologic abnormalities found at the time of admission were likely the result of the seizures. The initial acid-base abnormality was respiratory alkalosis without compensation. However, as seizure activity progressed and lactic acidosis developed, the dog's acid-base status included mixed respiratory alkalosis and metabolic acidosis. Hyperthermia and resulting mild dehydration were the most likely cause of the dog's high creatinine kinase activity, hyperphosphatemia, and hyperalbuminemia. Low serum globulin concentration and high alkaline phosphatase activity are likely explained by the dog's age. The other clinicopathologic abnormalities can be attributed to nutrient deficiencies of the homemade diet, including the determination of hyponatremia, hypochloremia, and hypocalcemia.

Dogs typically compensate for low amounts of calcium in the diet through integrated actions of parathyroid hormone, vitamin D, and calcitonin. The severe hypocalcemia and radiographically evident bone changes were likely a result of the low dietary amounts of calcium, vitamin D, and phosphorus and the calcium-to-phosphorus ratio of the diet (0.7). The low dietary amount of calcium was particularly deleterious in this young dog because calcium requirement is highest during growth. This dog was eating only 27% of the AAFCO minimum requirements for calcium for growth in dogs, although results of previous studies[2,13,14] suggest that large-breed puppies are even more susceptible to harmful effects of low-calcium diets during growth than are puppies of smaller breeds.

Nutritional secondary hyperparathyroidism typically arises from low dietary amounts of calcium and low or recommended dietary amounts of phosphorus. Although serum calcium concentrations are initially maintained in nutritional secondary hyperparathyroidism, hypocalcemia can develop once calcium is depleted from bone. Parathyroid hormone is released in response to the low blood ionized calcium concentrations, causing bone resorption and increased production of calcitriol from the kidneys. As a result, a high serum iPTH concentration would be expected in nutritional secondary hyperparathyroidism but was not seen in this dog, possibly due to IV administration of calcium gluconate prior to collection of blood samples for determination of iPTH concentration. The half-life of parathyroid hormone is < 30 minutes in people and decreases in response to calcium administration.[11] Also in nutritional secondary hyperparathyroidism, serum phosphorus concentration is usually low or within reference limits but was mildly high at initial analysis in the dog of the present report. Serum phosphorus concentration did return to within reference limits on day 3 of hospitalization. Conversely, vitamin D concentration is typically high in nutritional secondary hyperparathyroidism but was low in the dog of the present report, possibly due to the concurrent severe dietary deficiency of vitamin D. Another possible cause for the low serum vitamin D concentration is severe intestinal disease resulting in malabsorption of nutrients, although this puppy had no gastrointestinal signs for several months prior to referral. Because vitamin D is involved in calcium absorption in the gastrointestinal tract, signs of vitamin D deficiency overlap with those of calcium and phosphorus deficiency, including slowed growth, defective skeletal mineralization, pathological fractures, and, rarely, type I vitamin D–dependent rickets.[15,16]

Typically, severe chronic calcium deficiency results in diffuse osteopenia that is radiographically evident. In a recent report,[17] diffuse osteopenia involving the vertebral column, mandible, and scapula was described in a puppy fed a homemade premix and raw beef diet. In another report[18] involving dietary deficiency of calcium and phosphorus in a growing dog, diffuse osteopenia of the long bones was described. In the dog of the present report, long bone demineralization was not detected on radiographs of the forelimb at initial evaluation but was apparent on the radiographs of the dog's mandible. In most species, bone loss during hypocalcemia occurs first in the mandible, followed by cranial bones, ribs, vertebrae, and long bones.[19] The alveolar bone of the mandible is often first affected because this type of bone has the highest rate of renewal.[19]

In addition to calcium, vitamin D, and phosphorus deficiencies, the homemade diet fed to the dog of the present report also was deficient in a variety of other nutrients. Nearly all nutrients found to be deficient in the diet were below the minimum requirements of both the AAFCO and NRC,[2] although 2 (ie, tryptophan and zinc) were only below the minimum requirements of the AAFCO. Although the dog did not have obvious signs of other deficiencies, such as ophthalmic le-

sions from vitamin A deficiency or anemia from iron deficiency, deficiencies may have been identified in the dog if more sensitive measures, such as serum vitamin A concentrations or total iron-binding capacity, were assessed. However, these additional tests were not feasible because of financial constraints.

An additional nutrient that was of concern on the basis of the evaluation of the homemade diet was taurine. Taurine is not considered to be an essential nutrient in dogs, but taurine deficiency–associated dilated cardiomyopathy has been identified in some dogs, especially of certain breeds.[4–7] Many of the breeds considered at higher risk are large and giant breeds, including Saint Bernards.[4–7] Therefore, plasma and whole blood taurine concentrations were assessed because of the low dietary concentrations (compared with dietary taurine requirements in cats) in conjunction with the signalment of the dog. Both plasma and whole blood taurine concentrations were low at the time of admission. Dietary supplementation with taurine was initiated, and at the same time, dietary intake of taurine also was substantially increased. The diet also contained concentrations of methionine and cysteine that met the minimum requirements of the AAFCO and NRC, in which case dogs should be able to endogenously synthesize adequate taurine. Whether dietary supplementation with taurine was required is unclear, but given the low blood concentrations and possible deleterious effects of deficiency (and unlikely adverse effects), dietary supplementation with taurine was thought to be warranted. The taurine supplement was chosen on the basis of results of a study[20] investigating quality control and disintegration properties of commercial taurine supplements.

There were a number of nutrients to consider in selecting the optimal diet for this growing dog with multiple nutritional deficiencies. These included a diet that was nutritionally balanced and one with increased concentrations of calcium. Although serum calcium concentrations had returned to within reference limits before hospital discharge, restoring whole body stores and bone mineral content could take several months.[17] The fish-based commercial diet selected contained 4.5 g of calcium/1,000 kcal (1.92% calcium on a dry-matter basis). Although some authors recommend lower calcium intake for growing large- and giant-breed dogs to reduce the risk of developmental orthopedic diseases (0.7% to 1.2% dry-matter basis),[21] the diet was chosen on the basis of the severity of the dog's calcium deficiency, results of studies[3,14] on normal growth when feeding medium- to large-breed puppies diets with 3.9 to 5.7 g of calcium/1,000 kcal, and the NRC's safe upper limit for calcium of 4.5 g/1,000 kcal. Excess calcium is only 1 risk factor for developmental orthopedic disease in large-breed puppies, with rapid growth, excessive biomechanical forces, and genetics also playing pivotal roles.[22–25] In the dog of the present report, growth was carefully controlled with frequent assessment of weight and BCS, with adjustment of calories.

Another reason for the diet selected in the treatment of the dog of the present report was the purported history of a food allergy. Although it was explained to the owner that a food allergy was an unlikely cause of the dog's diarrhea as a young puppy (other possible causes included stress of transport, infectious causes, or other dietary properties such as fat), the owner was still highly concerned with feeding a diet containing chicken. The clinicians were concerned that if the possibility of a food allergy were not considered, the owner may have returned to an unbalanced homemade diet again if diarrhea returned on the recommended diet. In addition, it was possible that the dog had a food allergy to chicken, considering that up to one-third of food allergies develop in the first year of life, and chicken is reported to be one of the common allergens in dogs.[26,27] If a food allergy did exist in this dog, there were also a variety of ingredients other than chicken, on the basis of this dog's early diet, that could have been allergens, such as rice, wheat, fish, corn, or egg (although given that the homemade diet contained rice and egg, these were considered to be unlikely allergens). Confirmation of an adverse food reaction would have required an exclusive elimination diet trial for 8 to 10 weeks while feeding a novel or hydrolyzed protein diet, followed by reexposure to the allergen with return of signs.[27] With all of these issues to consider and after careful discussion with the owner, the fish-based commercial diet was selected. Although not a true elimination diet trial because it contained rice and fish, the diet chosen was otherwise novel, avoiding chicken, corn, and wheat that had been present in the chicken-based adult dry dog food. Although there are a small number of commercial novel protein or hydrolyzed diets that meet the dietary growth requirements, the fish-based commercial diet was chosen over these diets because it provided the highest amounts of nutrients of concern for the dog of the present report (eg, calcium, phosphorus, vitamin D, and taurine) and it also more closely met AAFCO requirements for growth.

Cooked meat and rice diets (eg, chicken and rice or hamburger and rice [other carbohydrate sources such as potato may be used in place of rice]) are commonly recommended by veterinarians for the temporary feeding of dogs with gastrointestinal abnormalities. Unless homemade diets are very carefully formulated (even when other foods or dietary supplements are added, as was done in this case), they are typically extremely nutritionally unbalanced. Feeding these diets for a few days is unlikely to induce harm. In an adult dog, even severe deficiencies do not often manifest themselves in obvious clinical signs unless used long term. However, in growing puppies, particularly a large-breed puppy as in this case, the dietary imbalances can induce severe clinical signs. This highlights the importance of obtaining a thorough dietary history and making specific recommendations to the owner regarding diet for both healthy animals and animals that require a modified diet because of gastrointestinal problems or other medical conditions. When owners insist on feeding a homemade diet, veterinarians should work with a board-certified veterinary nutritionist to formulate a balanced homemade recipe, formulate a plan for monitoring the animal, and ensure that the owner continues to follow the recipe. Also, given the unique requirements of growing animals (ie, all dogs < 12 to 16 months, depending on size) and their sensitivity to imbalances, homemade

diets, commercial foods that are not complete and balanced, or commercial foods that do not meet requirements for growth should be avoided in this group.

a. Purina Proplan Chicken and Rice Adult Dry, Nestlé Purina PetCare, St Louis, Mo.
b. Nupro All Natural Dog Supplement, Nutri-Pet Research Inc, Manalapan, NJ.
c. CVS calcium carbonate, 1,250-mg tablets, CVS Pharmacy, Woonsocket, RI.
d. Calcitriol capsules, TEVA Pharmaceuticals, Sellersville, Pa.
e. Taurine tablets, PetAg Inc, Hampshire, Ill.
f. Purina Proplan Chicken and Rice Puppy Canned, Nestlé Purina PetCare, St Louis, Mo.
g. Purina Veterinary Diets DRM Canine Dry, Nestlé Purina PetCare, St Louis, Mo.
h. Purina Gentle Snackers, Nestlé Purina PetCare, St Louis, Mo.

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