GENERALIZED OSTEOPENIA AND PATHOLOGICAL FRACTURES IN A PUPPY FED A RAW MEAT DIET

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Background

The popularity of feeding raw meat-based diets has significantly increased in the past few years but supportive studies regarding its advantages are still lacking. In fact, nutritional excesses or deficits are commonly observed and can be particularly concerning in young, growing animals. [1-2]

Nutritional recommendations for puppies take into consideration crucial nutritional factors, such as concentrations of calcium, vitamin D, phosphorus and calcium: phosphorus ratio [3,4]. Large breed dogs in growing phase are particularly predisposed to develop numerous orthopaedic and neurological diseases if fed unbalanced diets[3,4]. Nutritional secondary hyperparathyroidism is particularly important in immature, growing and lactating animals given their high calcium requirements for skeletal mineralization. [3-5]

This case report highlights clinical implications of feeding an unbalanced raw meat diet and accounts for the importance of owners' compliance whilst instituting a dietary plan.

Case report

A 5-month old male entire Bernese Mountain Dog was referred with a history of lethargy, intermittent lameness and marked difficulties in walking. The patient was first seen by the referring veterinarian 4 weeks prior to referral for occasional right pelvic limb lameness.

Symptomatic treatment with meloxicam was initially prescribed but no improvement observed. Radiographs of the stifles and pelvis performed at the primary veterinary practice raised the suspicion of panosteitis therefore meloxicam was withdrawn and a course of gabapentin (10 mg/kg TID) and prednisolone (1mg/kg SID) were prescribed. The puppy initially improved but over the next 2 weeks lameness started affecting all 4 limbs urging prompt referral.

The patient was fed a homemade diet consisting of a mixture of raw chicken and ground beef.

Orthopaedic and neurological examination were rather challenging due to marked osseous pain. The animal was unable to stand and displayed generally non-repeatable pain responses when the bones were palpated or the joints manipulated. The most significant pain responses were elicited on extension of both stifles and palpation of the thoracolumbar spine. The animal was still ambulatory but incredibly reluctant to do so and would vocalise on every attempt.

Serum biochemistry revealed moderate hyperphosphatemia, (2.31 mmol/L, reference interval 0.8–1.6) and increased serum alkaline phosphatase activity (ALKP) (228 U/L, reference interval 20–150). Serum ionised calcium was within normal limits (1.3 mmol/L, reference interval 1.15-1.33).

The plasma PTH concentration was found to be markedly elevated (633 pg/ml, reference interval 20-65 pg/mL) and Vitamin D concentration was low (56 nmol/L; reference interval 109-423). PTHrP was undetectable.

Radiographs of the stifles, antebrachium, lumbar spine and skull revealed a generalised osteopenia with the spinous processes of the vertebrae and wings of the ileum being the most severely affected (figure 1). There was an equivocal folding fracture of the right femoral diaphysis with no significant physeal, metaphyseal or epiphyseal abnormalities (Figure 2). Computed tomography (CT) confirmed marked disuse osteopenia with secondary pathological folding fractures in the left and right femurs and ischial bones (figure 3).

Based on the patient's clinical presentation and initial diagnostics the presumptive diagnosis **was nutritional secondary hyperparathyroidism.**

Medication prescribed by the referring veterinary surgeon was gradually reduced.

The puppy was hospitalized and treated with methadone 0.2 mg/kg every 4 hours intravenous (IV), paracetamol 10 mg/kg IV every 8 hours and strict cage combined with transition to a balanced growth formula.

Prior to discharge, 5 days after initial presentation, the animal was able to ambulate even though stiffness and kyphosis persisted. A re-evaluation appointment was scheduled in 4 weeks for repeated imaging and PTH essay. Unfortunately, the prescribed diet was discontinued at home and the patient was euthanized 3 weeks later given permanent skeletal deformities.

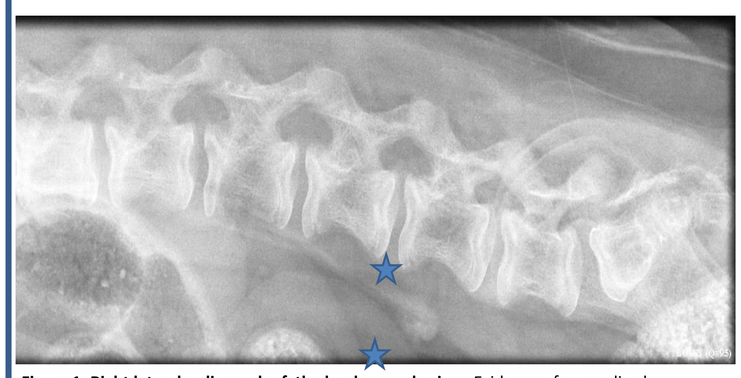

Figure 1. Right lateral radiograph of the lumbosacral spine. Evidence of generalized osteopenia of the vertebral bodies and spinal processes. (stars)

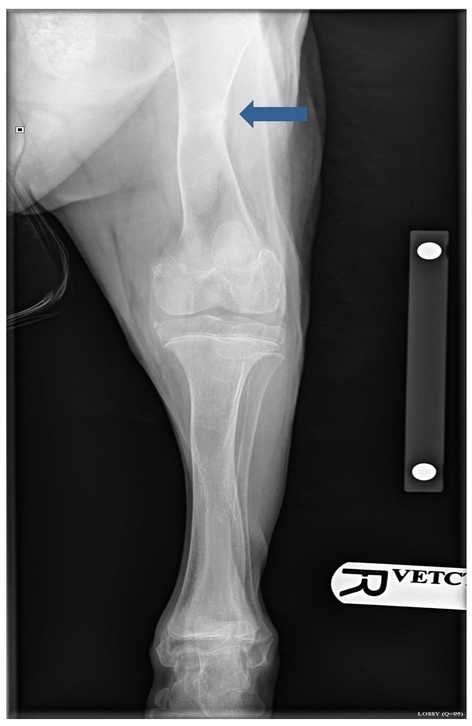

Figure 2. Craniocaudal view of the right stifle. Focal cortical irregularity and endosteal thickening y at the level of the mid distal femoral diaphysis suggestive of a folding fracture (arrow).

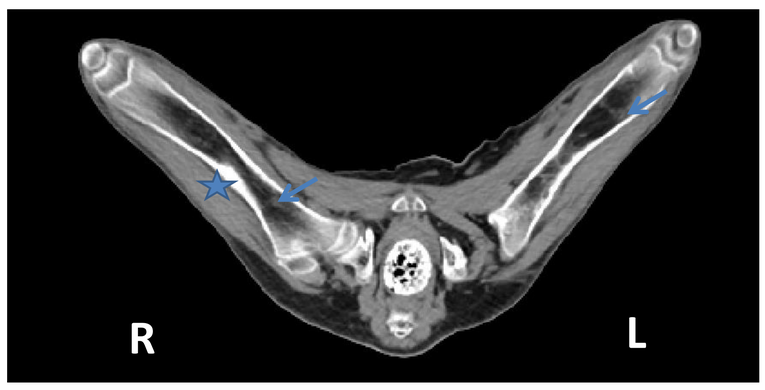

Figure 3. CT scan confirming generalized osteopenia.The medullary cavities are almost completely devoid of cancelleous bone/trabeculae (blue arrow**).** Periosteal and endosteal reaction in the mid to proximal diaphysis of the right femur corresponding to a jagged linear fracture line (star)

Discussion

The presence of diffuse bone pain and generalized osteopenia suggested a metabolic disease and a tentative diagnosis of secondary nutritional hyperparathyroidism (NSHP) was made based on dietary history, serum biochemistry abnormalities, hormone measurements and supportive imaging findings.

Dietary calcium insufficiency or hyperphosphatemia inducing hypocalcaemia are generally the most common causes of NSHP. Increased parathyroid activity can lead to compensatory normocalcemia via skeletal demineralization in contrast to what is expected in primary hyperparathyroidism and humoral hypercalcemia of malignancy. [5]

Radiological findings in this dog were consistent with reduced radiodensity of the skeleton and fractures of the long bones as seen in previous cases. [6-7]

The potential risks of providing a nutritionally incomplete and unbalanced diet, especially during a growth, cannot be overemphasized especially with recent trends in feeding home prepared raw diets. [1-2]

Recommendations on feeding raw meat based diets are typically based on opinion and peer reviewed studies are lacking[1-2.] In addition, most home prepared diets are deficient in 1 or several micronutrients resulting in medical conditions.

In conclusion, the case described is a contribution to the database of laboratory and clinical abnormalities identified in canine nutritional hyperparathyroidism. Successful treatment is attributed to prompt diagnosis, appropriate dietary management and owner compliance which unfortunately was not observed in this case.

References

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