

## Calcinosis circumscripta involving the metatarsal region in a dog with chronic renal failure

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A three-year-old intact female German Shepherd dog was referred to the Purdue University Veterinary Teaching Hospital for examination of a mass involving the soft tissues of the left lateral metatarsal area. The history was unremarkable. The cutaneous mass was firm with an ulcerated, exudative surface and measured  $6 \times 4 \times 2$  cm. Other findings on physical examination included small stature, poor body condition, and poor quality hair coat. The kidneys, as assessed by palpation, were small.

A complete blood count, serum biochemical analysis, and urinalysis was performed. A nonregenerative anemia (PVC 0.30 L/L) and lymphopenia ( $0.820 \times 10^9/L$ ) were present. Azotemia (serum urea nitrogen 38.6 mmol/L and creatinine 1644.2  $\mu\text{mol/L}$ ), hyperphosphatemia (3.6 mmol/L), hypercalcemia (3.28 mmol/L), and mild hyperamylasemia (1,236 IU/L) were present as well as mild hypokalemia (4.0 mmol/L) and hypochloremia (112.0 mmol/L). Urine was isosthenuric (1.010) with an inactive urine sediment, and urine culture produced no bacterial growth. Radiographs of the left metatarsus showed a multilobular, mineralized cutaneous mass that did not involve the local osseous structures or pad. Thoracic radiographs revealed a slight increase in interstitial pattern and mineralization of the major lobar bronchi. The kidneys were not visible on abdominal radiographs due to lack of contrast. Ultrasonographic evaluation revealed hyperechoic kidneys with thin cortices and dilated pelves.

Histological evaluation of a biopsy of the cutaneous mass revealed cystic structures that contained granular basophilic material surrounded by multinucleated giant cells, macrophages, and lymphocytes. Fibrous connective tissue was present between the cysts. A diagnosis of calcinosis circumscripta and chronic renal failure was made. The owner chose not to pursue any further

treatment and took the dog home to be euthanized by the referring veterinarian.

Calcinosis circumscripta, also known as calcium gout, apocrine cystic calcinosis, and Kalkgicht, is an uncommon skin disorder of dogs (1,2). It occurs mainly in healthy, young dogs of large breeds, particularly German Shepherds (3,4). In healthy dogs, lesions usually occur in the cutaneous tissues over pressure points and bony prominences, with the areas most commonly affected being the tarsometatarsal (24% of the reported cases), phalangeal (19%), elbow (17%), and cervical vertebral (10%) (3). In dogs with chronic renal failure, calcinosis circumscripta has only been reported to occur on the footpads (1,5,6). However, the case reported herein involved a young dog with chronic renal failure and calcinosis circumscripta associated with the soft tissues of the metatarsus. Hypercalcemia, hyperphosphatemia, and increased calcium-phosphorus product associated with the chronic renal failure likely caused or contributed to formation of the lesion. This case illustrates that dogs with chronic renal failure may have calcinosis circumscripta lesions associated with structures other than the footpads, and that dogs with calcinosis circumscripta should be evaluated for underlying renal disease regardless of their presenting signs.

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Calcinosis circumscripta lesions are usually nonpainful, multilobar masses that range in size from 0.5-10.0 cm (7). The masses can be fluctuant on palpation and, when incised, discharge a chalklike material. A diagnosis can be obtained by histological evaluation of the mass. The loculi contain amorphous material that is composed of calcium salts and cellular debris surrounded by macrophages, multinucleated giant cells, lymphocytes, and neutrophils (7). In general, the

histological character correlates with the chronicity of the lesion, and can be placed into three categories as follows: early lesions characterized by minimal inflammation; intermediate lesions having mild to moderate inflammation and mineralization; and late lesions characterized by marked granulomatous inflammation and mineralized amorphous material (3).

The cause of calcinosis circumscripta is still unknown. Concurrent illness is usually not associated with calcinosis circumscripta; however, early reports documented an association between chronic renal failure and calcinosis circumscripta of the footpads in six dogs (1,5,6). Different mechanisms have been proposed to explain the development of the lesion in dogs either without or with renal failure. Mechanisms for dogs without renal failure include the "trauma theory" (2,7). This mechanism may explain the occurrences of calcinosis circumscripta in large dogs with excessive, chronic abrasion of the tissue over the bony prominences, which may lead to granulomatous changes and dystrophic calcification in the dermis (3,7). Cystic apocrine glands may also develop calcinosis circumscripta from repeated trauma (2). Some dogs may have a hereditary predilection for the development of calcinosis circumscripta, since lesions have occasionally been reported in closely related dogs (8). Excessive dietary supplementation of vitamin D or calcium may also cause the lesion (1). No single mechanism seems to be responsible for all of the reported cases.

Calcinosis circumscripta is hypothesized to occur in dogs with renal failure when the serum calcium-phosphorus product is greater than 70 and the tissues are susceptible to metastatic calcification (6). Increases in the calcium-phosphorus product in dogs with chronic renal failure can occur from increased serum phosphorus concentrations, increased serum calcium concentrations, or both (9). Hyperphosphatemia is most likely a result of a decrease in the glomerular filtration rate and a decrease in phosphorus excretion in the kidney due to the progressive loss of functioning nephrons (9). Hypercalcemia occurs in 5–10% of dogs with chronic renal failure and may be due to a combination of factors (10). It may develop as a result of decreased renal excretion of calcium, increased intestinal absorption of calcium due to an exaggerated response to vitamin D, an increase in complex calcium concentrations in the blood due to increases in blood citrate concentrations, or due to increases in parathyroid hormone concentrations as a result of excessive secretion and reduced tubular degradation of parathyroid hormone (tertiary hyperparathyroidism) (10). The development of hypercalcemia in our case may have contributed to the formation of the calcinosis circumscripta lesion through its effect on the calcium-phosphorus product or through a direct effect.

In one reported case of a dog with chronic renal failure and calcinosis circumscripta of the footpad, hyperphosphatemia, normocalcemia, and a calcium-phosphorus product greater than 70 were present (6). The two other reports of dogs with chronic renal failure and calcinosis circumscripta of the footpads did not document serum biochemistry values (1,5). In our case, hyperphosphatemia, hypercalcemia, and a calcium-phosphorus product greater than 70 were observed and most likely contributed to the formation of the calcinosis circumscripta lesion. Other proposed causes of calcinosis circumscripta, such as hereditary predilection, vitamin supplementation, or repeated trauma to the leg, could not be identified, although this breed may be predisposed to the development of the lesion.

It is not known why the footpads may develop more severe calcification than the visceral organs in dogs with calcinosis circumscripta. The constant trauma to the footpads from walking and running may increase the susceptibility of the footpads to metastatic mineralization (5,6). Other mechanisms may also exist. Our dog showed evidence of bronchial calcification on thoracic radiographs in addition to the calcinosis circumscripta lesion.

Our case emphasizes the fact that dogs with chronic renal failure can have calcinosis circumscripta lesions located on the cutaneous tissues as well as on the footpads. This illustrates the importance of evaluating for underlying renal diseases in all cases of calcinosis circumscripta.

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