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CASE REPORTS

Calcium Pyrophosphate Deposition (Chondrocalcinosis) in a Dog

M. HEIMANN, J. L. CARPENTER, AND P. B. HALVERSON

Key words: Calcium pyrophosphate dihydrate crystal; chondrocalcinosis; dog; pseudogout.

Chondrocalcinosis (pseudogout) has been well studied in human beings but has only been reported in one dog.[2] The disease is characterized in human beings by deposition of calcium pyrophosphate dihydrate (CPPD) crystals in fibro- and hyaline cartilages and juxta-articular connective tissues involving one or multiple joints.[4–7] Larger joints are most often affected, and there is an increased concentration of inorganic pyrophosphate with or without crystals in synovial fluid. The patient may have recurrent attacks separated by asymptomatic intervals or may progress through longer attacks to chronic degenerative arthropathy.[4] Chondrocalcinosis has been associated with the following: knee trauma, meniscectomy, neuropathic joints, gout, hypomagnesemia, hypophosphatemia, familial hypocalciuric hypercalcemia, hemosiderosis, hemochromatosis, ochronosis, Wilson's disease, hypothyroidism, hyperparathyroidism, amyloidosis, and diabetes mellitus.[3,6,7] Familial examples have been observed with autosomal dominance mode of inheritance.[5] This paper reports chondrocalcinosis in a dog with clinical signs that suggest hypothyroidism.

A 15-year-old, 19.1 kg, castrated male English Pointer mixed-breed dog had a pericarpal mass that grew slowly for 4 years and caused mild but progressive lameness. During the last year, the growth enlarged by a third. The mass was subcutaneous and fixed to the deeper tissues. It was hard, multinodular, and surrounded the entire right carpus.

Radiographically, tightly aggregated or lobulated radiodensities were interpreted as mineralized soft tissue or bone (Fig. 1). The lesion surrounded the distal radius, distal ulna, and carpal joints.

Complete blood count and serum chemistry profile analyses were normal except for increases in amylase (1,379 U/liter; reference value, 440–1095) and cholesterol (517 mg/dl; reference value, 109–293).

The owner requested that the dog be euthanatized because of the inoperable forelimb mass, recently acquired fecal incontinence, persistent head tilt that was caused by a stroke 2 years earlier, seizure onset, and advanced age. A necropsy was performed.

The forelimb lesion was white, hard, lobulated, well-circumscribed, and encapsulated. When viewed on cut-surfaces, the confluent masses were adherent to joint capsules but did not involve joint cavities or articular surfaces. The nodules were white, chalky, granular, and separated by white to tan fibrous septa. Microscopic examination of a cytologic preparation obtained by scraping the granular substance disclosed numerous, variably sized rhomboid crystals both free and within macrophages, as well as a lesser number of needle-shaped crystals. Both crystal forms were weakly birefringent upon plane polarization.

Light microscopy revealed that the nodules were composed of pale-brown crystalline deposits separated by highly cellular fibroconnective tissue containing foci of metaplastic fibro- and hyaline cartilages (Figs. 2, 3). Foci of osseous metaplasia were rare and widely scattered. Microsections of tissue decalcified in 5% nitric acid were free of crystals. Microscopic examination confirmed the lack of involvement of joint cavities and also revealed that tendon synovial cavities were free of proliferative tissue and crystals (Fig. 2). Granulomatous inflammation with numerous foreign body giant cells infiltrated the septa and fibrous capsules of the nodules. Perivascular lymphocytic and plasmacytic aggregates were scattered in the surrounding loose areolar tissues. Mild, multifocal, synovial cell proliferation in carpal joints and tendon sheaths was noted.

Crystals were examined by alizarin red staining, plane polarization, scanning electron microscopy, energy dispersive analysis, and X-ray diffraction studies.[1] The weakly birefringent, rhomboidal, and rectangular crystals were alizarin red positive and similar to CPPD (Fig. 4). The calcium to phosphorus ratio was 1.04 with a standard deviation of 0.05 based on six measurements by X-ray energy dispersive analysis. The crystals were 60% monoclinic-CPPD and 40% triclinic-CPPD by X-ray diffraction.

The following findings suggest hypothyroidism: hypercholesterolemia; atrophy of epidermis, secondary hair follicles, and skin glands; atherosclerosis of the basilar artery; pale eosinophilic and occasionally mineralized thyroid colloid; and thyroid follicular cell lipofuscinosis. Other histologic thyroid abnormalities were high columnar-type follicular cell hypertrophy and scattered small nodules of follicular cell hyperplasia and hypertrophy that lacked colloid. The brown thyroid lobes were not atrophic and were 2.6 × 1.0 × 0.5 cm. Light microscopy also disclosed reactive lymphocytic follicles, plasmacytosis, and hemosiderosis of the right axillary lymph node and intramedullary pheochromocytoma of one adrenal gland.

This case of chondrocalcinosis in a dog is similar to the

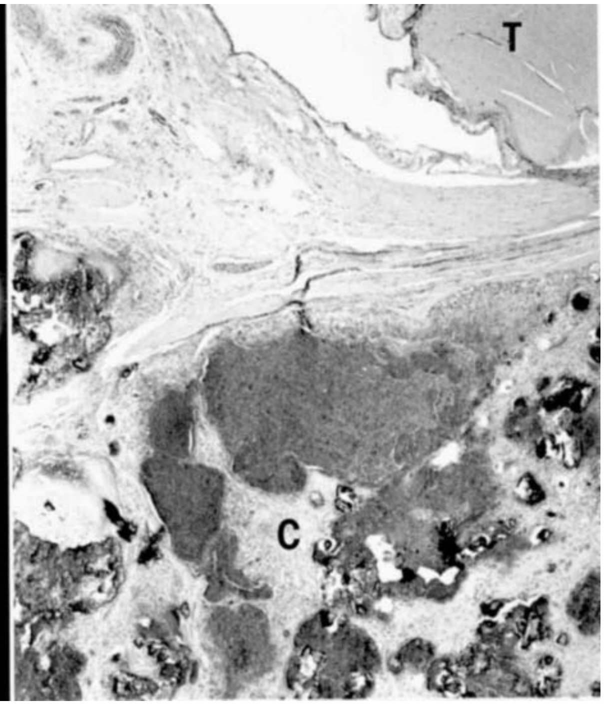
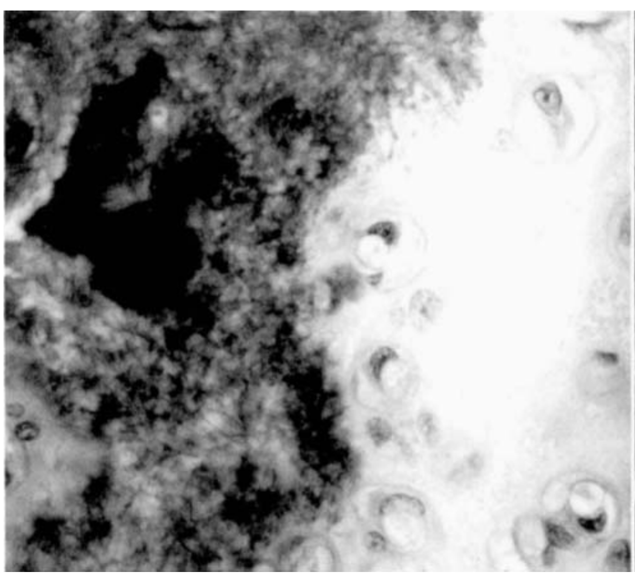


Fig. 1. Carpus, chondrocalcinosis. The lateral view reveals confluent, variably sized, radiodense, crystalline masses with many internal fine and irregular radiolucencies that represent trabeculae of collagen and metaplastic cartilage.

Fig. 2. Carpus, chondrocalcinosis. Dark variably sized and shaped deposits of calcium pyrophosphate dihydrate crystals are divided by irregular anastomosing trabeculae of fibrous and cartilaginous tissues (C). A tendon (T) and its synovial lining and cavity are not directly involved with chondrocalcinosis. HE.

only other reported case:[2] both dogs were old (11 and 13 years) at the onset of the lesion; the condition involved only one joint region in each dog (carpal and metatarsophalangeal); the growths were slowly progressive; both lesions had abundant periarticular cartilaginous metaplasia; and crystalline deposits did not involve articular cartilages or synovial spaces, indicating that the pathogenesis of the disease in dogs is not identical to that in human beings.[6]



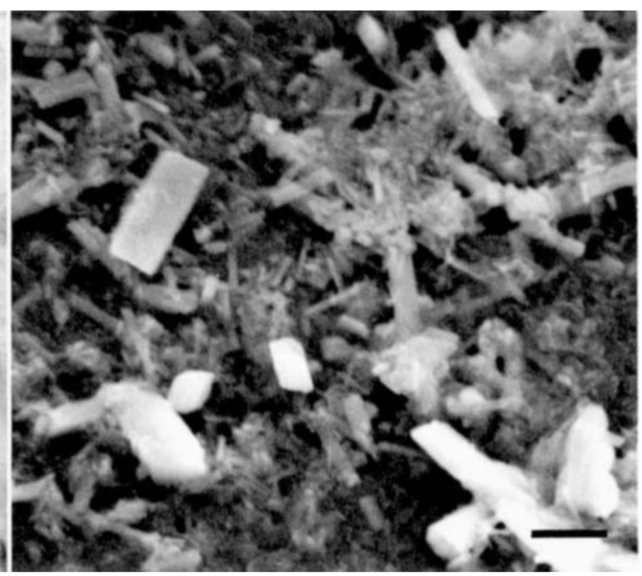


Fig. 3. Carpus, chondrocalcinosis. Dark calcium pyrophosphate dihydrate crystals (left) are adjacent to and within the margin of metaplastic cartilage (right). HE.

Fig. 4. Scanning electron micrograph of calcium pyrophosphate dihydrate crystals. Small slender forms predominate with larger rhomboidal and rectangular forms in the foreground. Bar = 10 μm.

One difference between this dog and the previously reported case is that our case had some clinical findings seen in hypothyroidism.

Acknowledgement

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References

1 Cohen AS: Calcium pyrophosphate dihydrate crystal; calcium pyrophosphate crystal deposition disease. *In:* Laboratory Diagnostic Procedures in the Rheumatic Diseases, ed. Cohen AS, 3rd ed., pp. 23–24, 335. Grune & Stratton, Orlando, FL, 1985
2 Gibson JP, Roenigk WJ: Pseudogout in a dog. J Am Vet Med Assoc **161**:912–915, 1972
3 Halverson PB, McCarty DJ: Pattern of radiographic abnormality associated with basic calcium phosphate and calcium pyrophosphate dihydrate crystal deposition in knee. Ann Rheum Dis **45**:603–605, 1986
4 McCarty DJ: Arthritis associated with crystal containing calcium. Med Clin North Am **70**:437–454, 1986
5 Rodriguez-Valverde F, Zuniga M, Casanueva B: Hereditary articular chondrocalcinosis, clinical and genetic features in 13 pedigrees. Am J Med **84**:101–106, 1988
6 Ryan LM, McCarty DJ: Calcium pyrophosphate crystal depostion disease; pseudogout; articular chondrocalcinosis. *In:* Arthritis and Allied Conditions, ed. McCarty DJ, 11th ed., pp. 1711, 1727. Lea & Febiger, Philadelphia, PA, 1989
7 Spiegel TM: Calcium pyrophosphate disease. *In:* Practical Rheumatology, ed. Spiegel TM, 1st ed., pp. 75, 139, 140. Wiley & Sons, New York, NY, 1983

Request reprints from Dr. J. Carpenter, Angell Memorial Animal Hospital, 350 S Huntington Avenue, Boston, MA 02130 (USA).

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Cutaneous Mast Cell Tumor in a Great Horned Owl (*Bubo virginianus*)

D. E. SWAYNE AND S. E. WEISBRODE

Key words: Avian species; mast cell tumor; skin.

Mast cell tumors are apparently very rare in avian species. A literature search revealed only one report in a chicken.[5] The following is a case report of two cutaneous mast cell tumors in a great horned owl (*Bubo virginianus*).

At necropsy, an adult male, wild, great horned owl was emaciated, had lesions of nephrosis, visceral urate deposition, and absence of skin, subcutis, and muscle over the distal half of the right radius and ulna. Two spherical, pink to red, circumscribed dermal masses 7 mm in diameter were present adjacent to the right orbit and left auditory meatus. The masses were well demarcated and composed of round to oval cells 9 to 15 μm in diameter that were uniformly and densely dispersed within a sparse collagen matrix (Fig. 1). Cellular boundaries were usually distinct. The cytoplasm was abundant and eosinophilic. Granules that were moderately metachromatic were evident with toluidine blue and Giemsa stains (Fig. 1, inset). These granules were strongly positive with periodic acid-Schiff stain (PAS) and negative for peroxidase. Most cells contained a single, centrally placed, round, oval, or occasionally indented nucleus. The nuclei exhibited mild anisokaryosis and infrequently contained one or two small nucleoli. Binucleate cells were common. Mitotic figures were rare. Perivascular heterophils and hemosiderin-containing macrophages were seen. Small foci of necrotic cells were present. Formalin-fixed tissue from the post-mortem specimen was examined ultrastructurally. The cells were round to oval, occasionally elongated, and the plasma membrane was molded to fit the shape of adjacent collagen fibers or cells. The cytoplasm contained three to eight round mitochondria located in the perinuclear zone, numerous polyribosomes, sparse rough endoplasmic reticulum, rare Golgi complex, and numerous (70 to 120 in plane of section per cell) membrane-bound granules (Fig. 2). These granules had an average diameter of 520 nm (mean of 25 randomly selected granules) and were of variable electron density. Most granules were moderately electron dense with fine stippled or particulate internal structure (Fig. 2, inset), and many of these granules appeared to interconnect. The cell surface was occasionally thrown into folds or villi. No basal lamina was seen. The light microscopic, histochemical, and ultrastructural features of these neoplasms support a diagnosis of cutaneous mast cell tumors. A diagnosis of systemic mastocytosis was not evident from other tissues examined.

Mast cells have been identified in phylogenetically diverse species of birds. Mast cells are present in various locations in birds including the liver, heart, lung, kidney, spleen, oviduct, peritoneum, skeletal muscle, and skin, but mast cells are most abundant in the digestive tract and ovary.[2,3,7,8] The organ distribution and quantity of mast cells varies between different species of birds. Mast cells are most numerous in the chicken (*Gallus domesticus*) and duck (*Anas platyrhynchus*) and in lesser numbers in the turkey (*Meleagris gallopovo*), ring-necked pheasant (*Phasianus colchicus*), and Japanese quail (*Coturnix coturnix japonicum*).[2]