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Calcinosis Cutis, Calcium Circumscripta, & Canine Gout by Fred Lanting

The subject ailment is an imbalance in mineral assimilation resulting in abnormal deposits, sometimes between bones, often in layers of the skin or integument. Calcium deposits in the skin can be the result of injury, of metabolic changes, or of unknown factors. Since mineralization (calcium deposits) in skin can occur in a wide variety of unrelated diseases, a common thread among them is not easy to establish. One form of the condition is related to, or could be an early indication of, a canine version of the disorder which in humans is called Cushing's Disease, although many dogs will never develop the distended abdomen, susceptibility to hematomas and bruising, or over-pigmented, sparsely-coated skin. Indeed, that may be a sufficiently different disorder that it should be classified as a separate variety of hyperadrenocorticism or hyperglucocorticoidism.

In 1998, I wrote the first version of this paper as a response to requests for information by a British friend who phoned and said he was afraid he would lose his 18-month-old German Shepherd Dog, which had been limping badly on several limbs. England has a lower population than the United States does, both in humans and dogs, so it is natural for us to see more incidences of any particular health problem or genetic anomaly in the U.S.A. Also, his GSD is of the "Alsatian persuasion" (style dating back to the go-it-alone and anti-German breeding of the post-war era), and this style has been decreasing numerically in proportion to the "Germanic" or what I prefer to call the international style in the past decade or more. So it was not surprising that he had not heard of any cases, even though the veterinary literature includes work of the ailment in his own country. The dog's feet were extremely sore, and a whitish fluid exuded from the pads; it was analyzed and the diagnosis in the U.K. was "calcium circumscripta", which I thought I knew simply as "gout". Many years ago, my friend and HD mentor, Dr. John Bardens, told me about a remedy or treatment he had devised for what he then called gout, but for the life of me, I could not remember what the medication was, or what variety of gout he meant. I was on the road when the call from England came in, but when I returned I consulted some references and was convinced that it was not this type. I told my English friend it may have been considered "rare" by his vet in England, but would be "downgraded" to "uncommon" in the U.S.A.

According to Carroll Weiss, Director of the Study Group on Urinary Stones, Health & Research Committee, Dalmatian Club of America, the term "gout" in the U.S. (and within medical schools, hospitals, medical libraries, and medical dictionaries), is very specifically defined. It is the deposition within tissue of insoluble uric acid or purines (or metabolites) due to an inborn defect in the normal production of urine within the liver and kidneys. Within both human and canine health circles, it is traditionally treated and controlled with one of several treatments, especially an anti-urate drug, "Allopurinol". This drug would be totally inappropriate for conditions like calcinosis cutis that mimic true gout. It acts pharmacologically on the defect of abnormally producing insoluble uric acid and is indicated only for urate-induced gout, abnormal urinary crystals, or stones. Other conditions certainly resemble "gout" if minerals other than urates are deposited. But "gout" is exclusively reserved for urates and purines. Dalmatian owners will probably want to go to website for more on these disorders as seen in their breed. Thank you, Carroll. Gout in humans has often been the subject of cartoonist humor, and I used to read many examples in the "funny papers" of the 1940s and for many years after. Maggie may have had the only chance of keeping Jiggs from sneaking out for a night with the boys when he was suffering from gout. The history of this cartoon goes far back to the Elizabethan era, when the British cartoonist Hogarth had a series entitled

"Marriage a la Mode." It showed the husband with his gout-afflicted foot bandaged and on a cushioned stool. This was to be copied up to the modern era, in drawings and stage plays. However, as my son found out when he had a "bout with gout", it is most assuredly not funny to the sufferer. In this disorder, the insoluble uric acid or its chemical relations are deposited within tissues where, untreated, they can result in almost intolerable pain and even bone degeneration. The most common site for the deposits are probably localized areas in the foot where they can become visibly detected as tophi, which are deposits of uric acid (or its metabolites or salts, like urates) within the tissues about joints. Deposits of calcium carbonate and/or other calcium salts within tissues can resemble or even mimic tophi. Weiss says tophi look like bunions. The word comes from the Latin word for sandstone, which is what it probably looks and feels like.

Dalmatians suffer from true urate gout. It is rare but it also seems to be specific to this breed. More commonly seen is its urinary stone manifestation problem in the breed. Dalmatians are apparently the only breed of dog born with the defect in their kidneys and livers that can result in uric acid or "chemical relations" becoming insoluble and precipitating out in urine as crystals or "stones". Two other species have the same inborn defect: man and apes. In humans, more develop the familiar gout than develop urinary stones.

Weiss says that when humans do indeed have urinary stones, they form more in the kidneys than in the bladder — unlike dogs — where over 95% form in the lower urinary tract, where which is far more treatable and responsive to current non-surgical methods than if found in the kidneys. Parenthetically, this is the reason why the DCA Study Group prefers the term "Dalmatian urinary stones" rather than the incorrect, misleading, and more serious problem of "kidney stones." Urinary stones form more in canines than in humans. Breeds other than Dalmatians can form urate crystals/stones but not for the same inborn reasons, according to Weiss.

The main subject of this article, however, is the calcinosis seen in other breeds, more in certain ones. It can be either generalized (a few to several areas) or localized (one or two spots). Considered a tumor (which word could refer to a cancer, a nodule, a cyst, or an impacted gland), the condition when found in the skin is also known as Calcinosis Cutis, which means calcified skin. It is usually a non-neoplastic (benign, not cancerous) disorder there. Boxers and Boston Terriers are predisposed to it on the ear and cheek. Calcinosis cutis circumscripta in humans is most often seen as nodules in the skin of the extremities, especially the hands (scleroderma). In the canine, it seems to be more variable in location and manifestation, but still frequently in areas of increased wear, though most researchers now discount any idea that trauma has any significance. Treating the dog with drugs designed to fight hyperglucocorticoidism is helpful in many but not all of the varieties or locations. As described above, there are other crystal-related joint disorders referred to as gout or calcium pyrophosphate-dihydrate disease (pseudo-gout), and calcific peri-arthritis/tendinitis, which are managed by uric-acid-lowering medication. You may have met some people like my son, who have suffered from one of these. A diet lower in meat and certain other foods is usually prescribed.

Histologically, the canine calcinosis disorder often appears as an amorphous granular material with fibrous trabecula ("bone") cells and inflammation around it. As the lesion progresses, ulceration often occurs. Sometimes it starts or occurs at injection sites or where ears are cropped. If the calcinosis develops in injured tissue, it could be localized, in which case some have surmised it to be often associated with demodex, TB, staph infection, or granuloma caused by a foreign body such as grit or sand imbedded beneath the skin, or it could be connected with epidermoid cysts or malignant tumors. If it is localized, it could still be considered coincidental that it is found at wound sites. If it is widespread, it is probably due to either hyperglucocorticoidism (hyperadrenocorticism) or diabetes. If there is no apparent damage to tissues, and no abnormalities seen in blood hormone levels, the calcium salt deposits may likewise be either localized or generalized. In the above types of the disorder, serum calcium levels (amount of calcium compounds circulating in the blood and lymph) are not abnormal, as is the metabolism of calcium and phosphorus. If, however, the disorder metastasizes (travels from original location to others by means of abnormal cells being transported via the bloodstream), there have been seen abnormal calcium levels and in all of those cases, chronic kidney disease has been observed. According to Muller, Kirk, & Scott's

text on Small Animal Dermatology, "No therapy is beneficial" if it develops into the metastatic form. Considering all variations, we see such cutaneous mineralization in 40% of all dogs with hyperadrenocorticism. A telltale sign in the haircoat may be loss of hair, or hairs easily pulled out of the follicles.

Whether associated with glucocorticoid abnormality (sometimes found in puppies) or from an idiopathic (unknown, spontaneous) cause (in which case it almost always shows up after a year of age), Calcinosis cutis may take a year to clear it up, with drugs in the former cases or spontaneously in the latter. Since mineralization (calcium deposits) in skin can occur in a wide variety of unrelated diseases, a common thread among them is not easy to establish. Over-mineralization (too much calcium in the diet) should be considered a suspected factor in gout as well as in so many orthopedic disorders.

Atop the kidneys sit the adrenal glands (whence comes the word "adrenaline"), the cortex layer of which produces hormones known as corticosteroids. One of these hormones is glucocorticoid, which affects the metabolism of glucose, a form of sugar taken in or even manufactured by the body. If the body makes too much, it results in that imbalanced hormonal condition known as hyperglucocorticoidism or hyperadrenocorticism, and if this becomes severe, an imbalance in minerals also occurs. The calcinosis cutis could be widespread, appearing in any or all of the following: skin along the back, armpit, groin, flanks, over bony protuberances such as foot bones and vertebrae, and (reportedly) apocrine (sweat) glands. In the dog, these apocrine glands are found primarily in the tongue and pads, although a small amount of perspiration is possible in the rest of the skin; in humans, these glands are mainly in the area of hair follicles. Researchers have held differing ideas regarding the involvement, if any, of these glands. The renowned dermatologist Dr. Danny Scott, whom I profiled several years ago in my Dog World article, "Itch!", has discounted the involvement of apocrine gland origins. Similar glands are found elsewhere in the integument, such as merocrine sweat glands in the pads, where the secretion is more watery, although a small amount of perspiration is possible in the rest of the skin. In the dog, other similar glands are found in the tongue.

How does canine calcinosis come about? Well, etiologically speaking, it could be that there develops an abnormal breakdown of hydrocortisone in the genetically-predisposed dog or even from an almost entirely environmental cause, which leads to molecular structural changes in proteins such as collagen and elastin so that the tissue chemically attracts and binds calcium. Also there may be unseen mineralization in lungs, stomach wall, and skeletal muscles, where there may be tissue damage at a later time. A good argument for neutering an affected dog is that almost everything is "genetic" to some degree. There are references in the literature about this once-called-gout occurring in related dogs, such as Dr. L. N. Owen's 1967 article on Irish Wolfhounds in Volume 8 of Journal of Small Animal Practice, although you probably want to remember that there are different types, and that which occurs in the hock possibly could have a different heritability than in other locations. Drs. Scott and Buerger, in the Nov./Dec. 1988 volume of the Journal of the American Animal Hospital Association, "found no indication of familial occurrence" in their study of idiopathic calcinosis circumscripta.

One form seems connected with polyarthritis or HOD (see Canine Hip Dysplasia, by this author, currently out of print, with 2nd edition being worked on) but in these cases it goes away when those diseases associated with mineral imbalance or poor metabolism of calcium subside. Those cases usually appear near the shoulder blades and hip joints. When occurring over pressure points and bony prominences or bones close to the skin, nearly a quarter of the lesions are seen in the hock area, almost a fifth in the phalanges of the toes, about 17% in elbows, and 10% in the lower dorsal neck area. There is ten to twenty times more involvement in the tarsal-metatarsal (hock) area than in the footpad. The dogs with calcification of the "skin" in the pads possibly are exhibiting a different form, and since they limp, it is diagnosed faster than if gout appears elsewhere in the skin as plaques, nodules, or papules (bumps). Typically, a milky or chalky white liquid, often gritty or paste-like, can be expressed if the pad is lanced or sliced, and this was the beginning of the definitive diagnosis in the case of my English friend's dog. One of my vet-tech correspondents described the hock lesions in her breed (Wolfhounds) as being sometimes open and weeping, sometimes closed and cauliflower-like. Her advice was if it were not open or very painful, "ignore" it for six months, as they often diminish in size and even disappear without treatment. She also had one of her pups develop a lesion on its tongue, and having chosen to delay surgery, found that it had gone from large-marble size to pea-size in four months. We should not draw conclusions from

one type and apply them to others.

Some 80% of cases of localized idiopathic calcinosis cutis are in large breeds including many Great Danes and Irish Wolfhound; over 50% of affected dogs are German Shepherd Dogs. Most are less than two years of age when diagnosed, as was the British GSD, yet most cases show up after one year of age, so it is just when a show career might be starting that the problem does, too. It is usually a benign disorder, with normal serum calcium levels (amount of calcium compounds circulating in the blood and lymph). However, the disorder can be cancerous and with the metastatic form of the disease, these levels will be abnormal, as is the metabolism of calcium and phosphorus. If the calcinosis is widespread, it is probably due to hyperglucocorticoidism or diabetes or else it has already metastasized.

If there is no apparent damage to tissues, and even if no abnormalities are seen in blood hormone levels, the calcium salt deposits may likewise be either localized or generalized. The benign nodules are generally up to one-quarter inch in diameter and shaped like domes although frequently they lie under a layer or more of skin so their shape is not seen until removed. Typically, treatment for this form (rather than drugs aimed at the adrenal glands) involves cutting out the granular material, but this can be from disappointing to satisfactory, depending on the individual dog, the degree and type of lesion, and removing the whole lesion. There does not appear to be development of new lesions in the same place after successful (complete) surgical excision, and many dogs have gone well over eight years without recurrence in the same location. Treatment of the generalized forms still involves treating the underlying causes such as skeletal disease or blood chemistry and metabolism. This may take a year to clear up, and that is about the same time that it takes for most cases to spontaneously regress. Generalized gout, whether associated with glucocorticoid abnormality (sometimes found in puppies) or from an idiopathic (unknown) cause almost always shows up after a year of age, and it may take a year to clear it up, with drugs in the former cases or surgically or spontaneously in the latter.

Some time after I faxed copies of medical articles to England, my friend informed me that his dog was being successfully treated and I later learned that it recovered completely, thanks to the surgical removal of the deposits. Although foot pad involvement in other breeds may indicate the metastatic variety, localized calcinosis circumscripta and successful surgical removal has been reported several times now in the German Shepherd Dog, and in breeds less often afflicted.

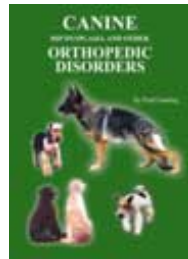
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Fred Lanting is an internationally respected show judge, approved by many registries as an all-breed judge, has judged numerous countries' Sieger Shows and Landesgruppen events, and has many years experience with SV. He presents seminars and consults worldwide on such topics as Gait-&-Structure, HD and Other Orthopedic Disorders, Anatomy, Training Techniques, and The GSD. He conducts annual non-profit sightseeing tours of Europe, centered on the Sieger Show (biggest breed show in the world) and BSP. Check out his website: www.MrGSD.com



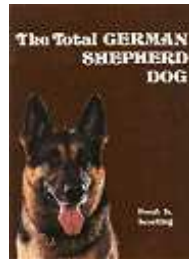
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Canine HD and Other Orthopedic Disorders by Fred Lanting.



It covers all joints plus many bone disorders and includes genetics, diagnostic methods, treatment options, and the role that environment plays. This new Hip Dysplasia and Other Canine Orthopedic Disorders\' book is a comprehensive (nearly 600 pages!), amply illustrated, annotated, monumental work that is suitable as a coffee-table book, reference work for breeders and vets, and a study adjunct for veterinary students, for the dog trainer and the general dog owner of any breed.

The Total German Shepherd Dog by Fred Lanting



This is the expanded and enlarged second edition, a "must" for every true GSD lover. It is an excellent alternative to the "genetic history" by Willis, but less technical and therefore suitable for the novice, yet very detailed to be indispensable for the reputable GSD breeder. Chapters include: History and Origins, Modern Bloodlines, The Standard, Anatomy, The German Shepherd in Motion, Shows, Showing, and Training, The Winners, Nutrition and Feeding, General Care and Information, Health and First Aid, Parasites and Immunity, Diseases and Disorders, The Geriatric German Shepherd, Breeding, Basics of Genetics, Reproduction, Whelping, The First Three Weeks, Four to Twelve Weeks, Trouble-shooting Guide

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