

Diagnosing and Managing Non-Pruritic Alopecia in Dogs

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Alopecia in the dog is a common clinical finding. It is most commonly associated with pruritus due to allergic skin disease. There are also many nonpruritic causes of alopecia. Since the skin and hair can only “react” in a limited manner regardless of the triggering event, signalment, history (hx), physical exam (PE) and laboratory testing (eg skin scrapings, skin biopsies, fungal cultures, endocrine testing, intradermal testing, etc) will be needed to help determine the underlying cause.

Once congenital, pruritic or infectious causes of focal to multifocal alopecia have been eliminated as a cause, the remaining alopecic diseases are associated with inflammation or interface dermatitis. Note that the inflammation may only be histologically apparent, not clinically observable. These can only be differentiated based on microscopic examination of skin biopsies. Vaccine induced alopecia is an example of an inflammatory alopecic disease. When performing a skin biopsy in an alopecic disease it is best to submit an elliptical shaped sample that has the tip of one end in the alopecic region and the other tip in the normally haired area. Be sure to request that the sample is sectioned from tip to tip (longitudinally) rather than transversely. This will allow the pathologist to see the progression of the lesion from early to late stages all on one sample.

Vaccine induced alopecia is most commonly associated with rabies vaccination and occurs due to ischemic changes in the skin. The alopecia occurs 2-12 months after administering a rabies vaccine. Small white breeds of dogs seem to be at risk for developing these lesions. SQ or IM injections have no impact on the occurrence of this reaction. Lesions consist of scaling, focal (occasionally multifocal) areas of alopecia, plaques, hyperpigmentation, nodules, erosions, crusts and cutaneous atrophy (scarring). The lesions may also develop at sites distant from the vaccination site. Histologically in addition to typical vasculitis changes, septal panniculitis and focal lymphoid nodules will be seen. Rule-outs are fairly limited but should include demodicosis, dermatophytosis, allergic skin disease and bacterial skin disease.

Dermatomyositis is an ischemic genodermatosis in collies and shelties involving both the skin and muscles. When it occurs spontaneously in adult dogs of other breeds there is only skin involvement. The onset of clinical disease in the inherited form is between 6 weeks and 1 year of age- usually occurring before 6 months of age. The lesions may be fairly limited and heal as the puppy matures or they may progress. Usually the lesions stop progressing by the time the dog is a year old. The cutaneous lesions, which are usually the predominant clinical sign, include focal to multifocal areas of alopecia, scaling, crusts, erosions, ulcers, depigmentation, hyperpigmentation and scarring. These lesions occur on the face, mucocutaneous junctions, carpal and tarsal regions and the tip of the tail and ears. Onychodystrophy may also be present. Secondary bacterial pyoderma may occur. Muscle involvement, which only occurs in the inherited form, tends to be proportional to the severity of the skin lesions and is usually identified subsequent to the cutaneous lesions developing. These dogs may develop megaesophagus or muscle atrophy involving the muscles of mastication and ambulation. Differential diagnoses for the skin disease include demodicosis, dermatophytosis, superficial bacterial folliculitis, DLE, cutaneous drug reaction, erythema multiforme, vasculitis and epidermolysis bullosa simplex. In the author’s experience, puppies are mostly commonly presented with limited facial lesions that the breeder claims are wounds/scars from the other puppies or from a cat in the household. Diagnosis is based on signalment, physical examination and histopathologic changes consistent with a vasculopathy.

Treatment for ischemic skin diseases would include avoiding the trigger and various immunomodulating drugs. For vaccine-induced alopecia the treatment options include pentoxifylline or surgical excision of the affected area. Pentoxifylline is a methylxanthine derivative that increases RBC deformability and lowers blood viscosity thereby allowing for better blood flow through narrowed/edematous vessels. It also suppresses synthesis of proinflammatory cytokines such as IL-1, IL-4, IL-12 and TNF- α . Pentoxifylline is administered at 15 mg/kg tid. There may be a 30-90 day lag before full clinical response is seen.

Other treatment options for ischemic dermatopathy include tetracycline (or doxycycline) and niacinamide. These drugs are used with, not replacing pentoxifylline. Doxycycline and niacinamide (D/N) have various anti-inflammatory & immunomodulating properties. The dosage for niacinamide in dogs or cats <10 kg is 250 mg q 8 hours. For dogs >10kg - 500 mg of niacinamide q 8 hours are administered. Doxycycline is dosed at 2 mg/kg q 24 hrs. If there is a clinical response (may take 2-3 months) the treatment is decreased from tid, to bid to sid. Side effects are rare but include vomiting, anorexia, lethargy, diarrhea and elevated liver enzymes from niacinamide and hepatotoxicity from doxycycline.

If there are focal lesions that fail to respond to the previous treatment, topical glucocorticoids (GC) may be added. The topical products are applied bid until clinical remission (not to exceed 21 days) and then tapered slowly over the next few months. Be sure to have the owners wear gloves when applying these products. Please note that topical steroids may cause pu/pd/polyphagia. This sensitivity to steroids is quite variable and may occur in unexpected situations. Topical tacrolimus (0.1%) may be used in cases that fail to respond to topical steroids, the pet has side effects to the topical steroid or for the dog that needs long term topical treatment to control the disease.

If the disease is more widespread or fails to respond to the previous treatments, prednisone may be used. It is administered at 1 mg/kg bid for 14 days. The dog is rechecked every 14 days. If the disease is in remission, the dose is decreased 25% every 14 days. The author defines “remission” as the absence of any active lesions. **DON'T TAPER THE DOSE TOO QUICKLY.** The goal is to maintain the dog on 0.25 mg/# or less every other day. Another option for SEVERE cases would include azathioprine along with the oral GC. The initial dose of azathioprine is 2.2 mg/kg sid. Once remission is achieved, and the dog is either off of GC or the lowest dose of GC has been obtained, AZA is then tapered usually every 30-60 days. When tapering AZA, the author will decrease the frequency, not the dose of azathioprine, first decreasing it to every other day and then if the disease is still in remission, to every 72 hours. When using AZA, a CBC, platelet count and serum chemistry profile are performed every 14 days for 2 months, then q 30 days for 2 months then q 3 months for as long as the dog is on AZA. Potential adverse effects include anemia, leukopenia, thrombocytopenia, hypersensitivity reactions (especially of the liver) and/or pancreatitis.

Cyclosporine (Atopica®) may be effective in some cases of ischemic dermatopathy. Be sure to use modified cyclosporine (Atopica®) since unmodified CSA is not absorbed as well. The dosage is 5 mg/kg sid.

Sulfasalazine (SSZ) is a sulfa that has both anti-inflammatory and/or immunomodulatory properties due to its prostaglandin synthetase and leukotriene inhibition. In the past it has been used for the treatment of colitis but more recently it has been used for vasculitis. Side effects associated with this drug include anemia, KCS and hepatotoxicity so a CBC, serum chemistry profile and Schirmer tear test are performed every 14 days for 2 months, then q 30 days for 2 months then q 3 months for as long as the dog is on SSZ. The dose for SSZ is 20-50 mg/kg tid (maximum 1 gm/dose), usually beginning with 20-30 mg/kg tid. Once the disease is in remission, the dose is slowly tapered

Sebaceous adenitis (SA) is an inflammatory disease of the sebaceous glands. Some people will separate this disease into the granulomatous form (Standard Poodle form= SPf) that is seen in Standard Poodles, Akitas, Samoyeds, Old English and Belgian sheepdogs and the short coated breed form seen in the Vizsla, Weimeraners and Dachshunds. The author believes this later form is not sebaceous adenitis but rather part of the syndrome known as sterile granuloma/pyogranuloma syndrome (sterile periadnexal granulomatous dermatitis) and will not be discussed in this lecture.

A genetic basis has been identified in Standard Poodles and is believed to be an autosomal recessive trait. Both the spontaneous and genetic forms of the disease occur in young adult to middle aged dogs.

Clinically the dog with the SPf will have adherent white scaling, follicular waxy “casts”, and matted hair from the waxy scale, varying degrees of hypotrichosis (including alopecia) and a dull appearance to the hair coat. In Standard Poodles many of the remaining hairs lose their curls. Secondary bacterial folliculitis may be present and result in pruritus. SPf tends to begin on the dorsum, especially the head and then progress caudally and distally onto the extremities.

Early histopathologic changes that are found with the granulomatous form include a nodular granulomatous to pyogranulomatous reaction in the ischemic region of the hair follicle that is unilateral (sebaceous glands are unilateral), follicular and surface hyperkeratosis (clinically will appear as scaling). In the end stage of the disease, the inflammation has resolved and you will be left with perifollicular fibrosis, follicular atrophy and absence of sebaceous glands. Treatment for the SPf includes treating secondary bacterial or *Malassezia* infections. Pre-bath spraying with baby oil, bathing with a keratolytic shampoo (eg sulfur/salicylic acid containing product) and follow with a humectant. Keratolytic agents will **cause desquamation of the cornified epithelium, basically** loosening the outer layer of the skin (SC). Oral omega 3/6 combination products at double the bottle dose and evening primrose oil (500 mg bid). The author has discontinued using oral Vitamin A for this disease. This is based on a studyⁱ that revealed there was no correlation between vitamin A dosage and response to treatment nor any difference between dogs responding and those not responding to adding vitamin A to topical therapy. In addition, there is evidence that retinoids are the most potent pharmacological inhibitor of sebum secretion. Histological changes in sebaceous gland size can be seen after 8 weeks of treatment. The sebaceous glands have a reduced size and the sebocytes appear undifferentiated with decreased lipid accumulation. These are undesirable effects in treating sebaceous adenitis.

In a study oral cyclosporine was used to treat 12 dogs with SA (not just SPf)ⁱⁱ. Ten of twelve dogs improved within 4 months however most needed topical therapy once the mCSA was discontinued. In summary, treatment with mCSA resulted in clinical improvement in dogs with SA, with the greatest improvement evident within 4 months after the initiation of treatment. The authors concluded that long-term treatment appears to be necessary to control the disease. The authors reported that there was some evidence that mCSA was of limited benefit in dogs with chronic disease in which the perifollicular inflammatory reaction had already resolved. Therefore, treatment with mCSA should be initiated as early as possible during the course of the disease.

A subsequent study is only available in abstract form so details are lacking. This study involved 20 dogs with SAⁱⁱⁱ. Initial therapy included essential fatty acid supplementation with a total gamma linolenic acid dose of 10–20 mg/kg once daily and an antiseborrheic shampoo twice weekly. All animals were assessed at 3 weeks. An improvement in coat condition was noted at this time, but there was no evidence of hair regrowth. Treatment was started with topical cyclosporine. Twenty-five millilitres of cyclosporine (Neoral oral solution, 100 mg/mL) made up to a total volume of 250 mL of liquid with sterile water (making it a 1% solution) was applied to the coat once daily followed by an emollient spray. At a 6-week recheck, further improvement was noted. In

some cases, new hair regrowth was apparent. In six dogs, blood samples were taken at 9 weeks to measure blood levels of cyclosporine. In no case could cyclosporine be detected. Therapy was successful in every case, but was deemed too labor intensive by the owners of some dogs. Despite good initial improvement in their dog's skin condition, they were lost to follow-up. In all other cases, once hair had regrown after 8–12 weeks, the frequency of application could be reduced to once or twice weekly.

In a study using 9 dogs Lucas et al used a 0.4% CSA solution^{iv}. He made the solution by mixing four 100 mg capsules in 100 mL vegetable oil. The solution was applied twice per week. Clinical improvement was noticed in all dogs, and total hair regrowth occurred in 4 months. Topical (0.4%) cyclosporine A applied twice a week was well tolerated and efficacious in the symptomatic treatment of sebaceous adenitis in dogs.

Lastly there was a study that revealed that the combination of topical treatment and oral CSA gave the best results^v. Differences between the treatment protocols are marginal. Topical treatment, both alone and in combination with CsA, appeared to reduce scaling more effectively than CsA alone. Both therapies reduced alopecia. In the study there was some evidence suggesting a synergistic benefit on both scaling and alopecia if both treatment options were combined. Inflammation of the sebaceous glands was reduced the most by a combination of both CsA and topical therapy. There was evidence that regeneration of sebaceous glands is best achieved by CsA, either given alone or in combination with topical treatment.

The next group of alopecic diseases that will be discussed are the ones that are diffuse or symmetrical on examination. The first group we will discuss are the endocrinopathies. Hypothyroidism is one of the most over-diagnosed endocrine disease in the author's referral practice. Hypothyroidism is most commonly caused by an immune mediated destruction of the thyroid gland. Middle-aged medium sized to large breed dogs are the most commonly affected dogs. Clinical findings that have been associated with hypothyroidism are quite extensive and will not be reviewed here. A few dermatologic clues would include seborrhea sicca or oleosa, poor hair regrowth (seems to be a more common complaint than spontaneous alopecia), recurrent bacterial pyoderma and a dry, dull hair coat. Alopecia (triangular in shape) just caudal to the nasal planum is another finding that suggests hypothyroidism. The "frizzies" may be seen in Golden retrievers and Irish setters. CBC, serum chemistry profile and urinalysis may reveal mild nonregenerative anemia, hypercholesterolemia and hypertriglyceridemia. Thyroid testing is needed for a definitive diagnosis of hypothyroidism. Thyroid tests that are of value include Total T4 (TT4), free T4 by equilibrium dialysis (fT4ed), thyroid stimulating hormone concentrations (cTSH), thyroglobulin autoantibody (TgAA), T4 autoantibodies (T4ab) and T3 autoantibodies (T3ab). Details of these tests sensitivity and specificity are beyond the scope of this lecture.

The thyroid profile requested by the author includes TT4, cTSH, TgAA, T4ab, T3ab. The author will have a fT4ed added to the profile if there are T4ab present, if non-thyroidal illness is present or the dog has received drugs known to affect the thyroid. In general DOGS MUST NOT HAVE RECEIVED TOPICAL OR ORAL STEROIDS FOR 30 DAYS OR REPOSITOL STEROIDS FOR 3 MONTHS BEFORE TESTING THE THYROID. Also, they must not have received sulfa drugs for at least 30 days. For dogs with hypothyroidism, after 1 month of therapy (L-thyroxine 0.02 mg/kg bid-use BRAND NAME ONLY), a blood sample is submitted 4-6 hours post pill for a TT4. The levels should be in the upper range of normal or even a little higher than normal.

A far more common endocrinopathy seen by the author is hyperadrenocorticism (HAC). It is not the purpose of this lecture to discuss all the symptoms of HAC but a few points must be made. In dermatology it is NOT uncommon to have a dog with HAC present without pu/pd or a potbelly appearance and may ONLY have a recurrent pyoderma, poor hair regrowth or non-inflammatory truncal alopecia. If there is a suspicion that the dog may have an endocrinopathy (based on PE, cbc, serum chemistry and urinalysis results) then it is important to first rule out HAC since a dog with HAC may have a low thyroid profile due to the influence that steroids have on the thyroid gland. The 2 screening tests that are used by the author are the ACTH stimulation and the LDDS. If the dog has a history of steroid exposure, then an ACTH stimulation test is performed. If the dog has no recent steroid exposure, then the author prefers to begin with a LDDS test. Note that 1 normal screening test doesn't rule out HAC. The author believes that the sensitivity of the LDDS is much better than the ACTH stimulation. Treatment for HAC is based on the severity of the clinical signs. Either trilostane or mitotane may be used for treatment.

Dyscyclic follicular diseases of unknown etiology (post clipping alopecia, alopecia X, seasonal flank alopecia.) are diseases in which the hair follicle is structurally normal but it is not cycling properly. Rule outs for these dyscyclic diseases include the endocrinopathies already discussed and also hyperestrogenism (sertoli cell tumor associated).

Alopecia X is a syndrome of unknown etiology. Theories abound as to the cause including an adrenal sex hormone imbalance, an abnormal metabolism of hormones by the hair follicle or a hormone receptor problem at the follicular level. The later theory is supported by the observation that hair regrows at the site of skin biopsies. This ability to induce hair regrowth by localized trauma would suggest a local inhibition of hair cycling rather than systemic. Alopecia X occurs in plush coated breeds and in poodles. It occurs in young adults of either sex or reproductive status. Clinically these dogs lose their guard hairs, beginning on the neck and progressing to the shoulders, trunk and thighs. Eventually the dog may have a woolly, cream color coat. In some dogs this may progress to alopecia with hyperpigmentation. Diagnosis is based on signalment, hx, PE and ruling out (r/o) other alopecic diseases. Histopathology can support but not diagnosis Alopecia X. That is because the findings with Alopecia X resembles other dyscyclic alopecic diseases such as hypothyroidism, hyperadrenocorticism, gonadal sex hormone abnormalities, recurrent flank alopecia and

post clipping alopecia. Histologically, these diseases are characterized by many specific (follicular atrophy, telogenization of follicles with excessive trichilemmal keratinization (flame follicles), orthokeratotic hyperkeratosis, follicular keratosis, sebaceous gland atrophy), but nondiagnostic (nondifferentiating) findings. An adrenal sex hormone panel stimulation test can be performed but it is of questionable value in the author's opinion. Treatments that have been used with variable success include neutering, sex hormone replacement (estrogen OR testosterone), low dose lysodren, melatonin, trilostane, growth hormone and thyroid supplementation. All of these treatments may cause a temporary improvement in the alopecia (nonspecific anagen induction?) but rarely is the hair coat returned to normal. Also these medications (other than melatonin) are associated with potentially significant side effects. In the author's practice, if a diagnosis of Alopecia X is made then the client is counseled about the choice in treating a cosmetic disease with potent drugs. Neutering is recommended if it is an intact animal. If the alopecia fails to respond to the neutering, a therapeutic trial with melatonin 3-6 mg tid for 90 days is performed.

Seasonal flank alopecia (SFA) is a nonscarring alopecia that has been reported in a variety of breeds, but it has been reported to be more common in Boxers, Airedales and Bulldogs. The etiology is unknown. Some people think that it is caused by a "melatonin deficiency" since many of the dogs develop the lesions in the fall, when melatonin levels should be increasing and some dogs respond to melatonin administration. But there are some cases that the hair is lost in the spring and regrows in the fall so it makes this etiology impossible. The disease occurs in young adult dogs and will begin most commonly in the fall with spontaneous resolution in the spring. This disease may occur once and never recur, it may recur each year with each episode involving larger areas of the body, or it can occur once and never completely resolve. The lesions involve the flanks and sometimes the caudal lateral thorax. The alopecia is usually bilateral with annular lesions that may coalesce into polycyclic lesions with hyperpigmented and smooth glistening skin. **Papules and pustules consistent with a bacterial pyoderma may develop in these areas.** Diagnosis is based on r/o other nonscarring alopecias – hx alone may be diagnostic if it is a recurrent problem. Biopsy can support but not diagnose SFA. Treatment is again either a tincture of time or melatonin. Since the disease usually goes into spontaneous resolution it may be difficult to determine if the melatonin had any impact, especially the first time the disease occurs. In the author's practice melatonin is more commonly used to prevent symptoms by beginning therapy just prior to the onset of the symptoms (if there is a seasonal pattern). Dose is as discussed previously.

Post clipping alopecia occurs primarily in the Arctic breeds. It has been theorized that these breeds have a very long telogen (resting) phase to their hair cycle in order to preserve a high protein substance (hair!). If the hair is clipped during the telogen stage, it will not regrow until it cycles back to the anagen stage. Others have suggested that when the hair is clipped there is decreased blood flow to the area (to minimize heat loss) leading to a decrease in growth factors. Diagnosis is based on hx and r/o endocrinopathies. Histopathology will reveal follicles of normal size but in most are in telogen. Treatment is tincture of time or sometimes a 7-10 days course of thyroid supplementation (will stimulate anagen formation) or a 90 day trial of melatonin

The structural follicular dysplasias -color linked, non-colored linked and pattern baldness all have an abnormality not just of the hair follicle but also the hair shaft. Be aware that finding dysplastic hair follicles on histopathologic is not adequate evidence to diagnosis a structural follicular disease; there should also be dysplastic hair shafts. A study in 1998 reported that 46% of the dogs with an endocrine alopecia had dysplastic hair follicles but less than 1% had concurrent dysplastic hair shafts.

Colored linked alopecias include color dilution (mutant) alopecia (CDA) and black hair follicular dysplasia (BHFD). CDA occurs in dogs with a blue or fawn hair coat. These hair coat colors occur as a result of the effect of the "dilute" gene on black or brown hairs respectively. Any dog with a blue or fawn coat may be affected by CDA but not always. Dobermans and Great Danes are the most common breeds seen in the author's practice affected by CDA. A dog with this autosomal recessive genodermatosis is born with a normal coat but as the dog matures, usually beginning at between 4 months of age and 3 years, it will develop varying degrees of hypotrichosis (including frank alopecia) affecting the "dilute color" areas only. The hair coat will become dull and there will be scaling and comedone formation. Secondary bacterial pyodermas are frequently present. The exact cause of the hair shaft abnormality is not known but is believed to be related to a dysfunctional melanin transfer from the melanosomes to the hair matrix or a defect in the storage of the melanin once it is in the hair shaft. The result is melanin clumping. This clumping leads to weakening and eventual fracturing of the hair shaft. Diagnosis is based on hx, PE, appearance of hairs on a trichogram, r/o other alopecic diseases (especially demodex, dermatophytosis, bacterial pyoderma and endocrinopathies) and is supported by histopathology. Microscopic examination of plucked hairs will reveal melanin clumping in the hair shafts and disruption of the normal hair shaft architecture. Treatment (other than elimination from the breeding stock) is directed toward managing the secondary pyodermas and seborrhea. Bathing, humectants, fatty acids +/- antibiotics are the mainstay of therapy. Melatonin, which can stimulate hair cycling, has also been reported to improve hair coats in some dogs. The author uses melatonin, 6 mg tid, as a 90 day therapeutic trial.

BHFD is an alopecic disease of dogs with bicolored or tricolored hair coats such as Boston Terriers, Basset hounds and Cocker spaniels. It has been reported to be inherited as an autosomal recessive trait. This tardive disease is also believed to be due to a defective transfer of melanin leading to melanin clumping that weakens the hairs and eventual fracture. Usually abnormalities of the hair coat are noted by the time the dogs are weaned. Initially changes consist of a dull hair coat affecting only black hairs. Eventually these areas become alopecic. As with CDA secondary pyodermas may occur. It may be easiest to think of BHFD as a localized form

of CDA. Histopathology is similar to CDA and diagnosis is based on signalment, hx, PE, appearance of hairs on a trichogram and can be supported by histopathology. Treatment is the same as CDA.

Non-colored link follicular dysplasias have been reported in a number of breeds including Portuguese Water dogs, Irish Water Spaniels and Curly Coated Retrievers. Between 6 months and 6 yrs of age (depending on the breed) these dogs develop symmetrical hypotrichosis to alopecia usually beginning on the neck and progressing to the shoulders, trunk, tail and thighs. Any remaining truncal hairs may have a color change (lightening). In dogs, estrogen receptors are present in telogen hair follicles and are important in keeping hairs in this phase. In Irish Water Spaniels dietary change (avoiding soy which may contain phytoestrogens) has been reported to be effective. Melatonin and trilostane both block estrogen receptors and may account for the effectiveness of these drugs in a variety of canine alopecic diseases.

Pattern baldness alopecia (PBA) is also a tardive genodermatosis. The dogs are born with a normal coat but develop PBA at 6 months-1 yr of age. There are 4 different forms of this non-inflammatory, non-pruritic alopecia. One form occurs in male Dachshunds. These dogs develop a slowly progressive alopecia and hyperpigmentation of the pinnae. A second form occurs in primarily in female Dachshunds, Chihuahuas, Whippets, Manchester Terriers, Greyhounds, and Italian Greyhounds. This form is identical to the first form except for the distribution of the alopecia. In this form there is progressive alopecia caudal to and involving the pinnae, ventral neck, ventrum and caudomedial thighs. The 3rd form affects American Water Spaniels and Portuguese Water Dogs (see above). The last form is seen affecting the caudolateral thighs of Greyhounds. Regardless of the form of the PBA, diagnosis is made on signalment, hx, PE, ruling out other alopecic diseases and supported by histopathology in which there is miniaturization of hair follicles and shafts with normal adnexa. There have been reports of some dogs improving with melatonin.

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