Sebaceous Adenitis in the Poodle

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Sebaceous adenitis (SA) is a skin disease that affects many different breeds, including the poodle. It is considered to be a cosmetic disorder, and is generally not life-threatening if treated properly. Unfortunately, however, many owners of affected dogs may choose to euthanize the dog rather than deal with the condition and its side effects. The most common breeds it affects are the Akita, and the standard variety of the poodle. Other breeds which have also been diagnosed with SA include sporting breeds such as viszlas, weimeraners, and golden retrievers, as well as herding dogs such as the old English sheepdog and German shepherd dog, and toy breed dogs like the miniature pinscher. This skin condition has also been diagnosed in mixed breed dogs as well as purebred dogs. SA most frequently occurs in young dogs between 1-5 years of age, although it can occur both earlier and later in life. The disease is believed to be an autoimmune condition wherein the sebaceous glands located in the dermal layer of the skin become inflamed and eventually destroyed.

When the sebaceous glands are destroyed, the essential oils and lubrication (sebum) are lost and the dog's skin becomes dry and flaky. As more glands are lost, the hair follicle becomes affected and the dog will begin to have thinning hair that eventually falls out, leaving dry, scaly, hairless patches of skin. Severely affected dogs that are left untreated can lose the majority of the hair and the skin becomes very unhealthy. The dry, flaky skin has lost the lubrication and protection normally provided by the sebaceous glands, and now becomes brittle and often has an unpleasant odor. The skin can become cracked and irritated, resulting in open sores, poor wound healing, and increased risk of infection. Left untreated, the dog will become very uncomfortable and may develop severe, even life-threatening, infections.

While it is not difficult to control the symptoms of sebaceous adenitis, there is no cure for the disease. The main focus of treatment is to replace the lost sebum and restore the health and elasticity of the skin. This requires frequent exfoliating baths (weekly or every other week), baby oil soaks, and conditioning. If the owner is unable to bathe or groom the dog themselves, it can become an expensive process. Even with frequent bathing and conditioning, affected animals will often have heavier shedding of the hair coat, or in the case of the poodle will usually have "dandruff" present even in between baths. The hair coat will grow back with proper treatment, but the disease can wax and wane, resulting in times of thinning hair or increased hair loss in between times of healthier coat appearance. If the source of the autoimmune response is eventually characterized, it is hopeful that in the future more effective treatments will become available.

It is theorized that sebaceous adenitis is inherited as an autosomal recessive gene, with or without modifying genes. An autosomal recessive gene is one that requires that an individual receive two copies (one from each parent) of the defective gene in order to

express the disease. There are several groups researching SA at this time, with standard poodles, Portuguese water dogs, and other breeds. Sebaceous adenitis can skip generations and appear in varying levels of affectedness, from subclinical (no obvious changes in appearance of the coat and skin seen, but microscopic changes are evident) to severe (total loss of hair coat, scaly patches of skin, secondary infections, etc.). Because there can be varying levels of sebaceous adenitis symptoms, it is possible that multiple genes are involved in inheritance of this disease, controlling the expression of the autoimmune attacks on the sebaceous glands. In the past it was theorized SA was inherited as a simple autosomal recessive and that standard poodles may have a carrier rate as high as 50%, meaning that at least half of the standard poodle population have the ability to reproduce affected puppies. However, over time we have not seen a huge increase in the amount of SA affected puppies as would be expected in such a scenario, therefore either the carrier rate is less than 50%, or there are multiple genes involved in expression of the disease.

Currently the only way to definitively diagnose SA is though a skin punch biopsy evaluated by a dermatopathologist. The procedure involves applying a local anesthetic to the area to be biopsied (typically between the shoulder blades along the back), then obtaining a 6 mm punch biopsy of the site. This must be a full-thickness wedge of skin including the epidermal, dermal, and subcutaneous layers. The 6 mm full thickness wedge of skin in placed in formalin and sent to accepted laboratories for evaluation and/or registration. The categories for evaluation include subclinical, affected, and normal. Subclinical dogs have no external signs of the disease, their coat and skin appear normal, but the microscopic evidence shows inflammatory cells around the hair shaft and sebaceous glands. Affected dogs have clinical signs of the disease, including symptoms mentioned above, and the microscopic evidence will show inflammatory cells, loss of sebaceous glands, and damaged hair shafts with loss of hair. “Normal” dogs are only considered normal at the time of biopsy, as they can become subclinical or affected in the future. Dogs that are categorized as normal have no inflammatory cells associated with the sebaceous glands, and the hair shaft and surrounding tissue are microscopically normal.

At this time it is recommended that normal dogs be re-tested yearly or every other year with the punch biopsy method. Once a dog has been classified as subclinical or affected, it does not have to be tested again. In fact, once a dog has been classified as subclinical the laboratory will not evaluate future samples in the event that an owner is concerned about a misdiagnosis. This has caused some controversy in certain cases, as there have been dogs that have been diagnosed as subclinical and then later evaluated by board certified pathologists and been found to be normal. There is certainly some indication that other biological processes can confound the biopsy test. Occasionally a result will come back as "equivocal" and the dog is recommended to be retested at a later date or a different site and may then be found to be normal. Dogs with allergic dermatitis may have inflammatory cells present near the hair shaft, and damaged hair follicles from trauma and over reactive immune response can be confused with early SA resulting in an "equivocal" diagnosis. Other skin conditions, such as an inflammatory reaction to the application of flea and tick control or other parasite control ("spot-on" preventatives) can

mimic the inflammatory response of SA. One wonders if such scenarios may result in an incorrect subclinical diagnosis, which cannot be changed and will not be re-evaluated for normalcy.

There are many concerns about using the skin punch biopsy test as a determination for SA. The skin biopsy test itself is an invasive procedure, albeit a minor one, that requires time to heal and keep from becoming infected. When a poodle is in full show coat, the test can have a higher risk of infection, hair coat matting, and minor hair loss in that area (which is usually between the shoulder blades and the one area that should have the longest and thickest hair growth!). The SA test is a phenotypical test, meaning it is only evaluating the expression of the disease, and is not actually able to detect carriers for the disease versus clear and affected.

The biopsy test is also controversial concerning accuracy, given the fact that the test center will not re-evaluate at least the subclinical animals to determine if there was any underlying error or confounding condition that may have resulted in the subclinical result. Only equivocal results are invited for re-testing. It is standard for most laboratories to re-test results that appear to be contradictory to clinical symptoms. Laboratory error is always a source of misinterpretation in any test situation, just as labeling errors, owner misidentification, and other clerical and interpretive mistakes can be made. Why this standard is not upheld in SA testing is one of the questionable aspects of the test itself. Since re-testing is not available for subclinical cases, and since it is *possible* that an individual may be diagnosed as subclinical due to underlying causes other than SA, some breeders are choosing not to utilize the test as a screening tool. There are also those who feel that yearly skin biopsies are too invasive a procedure for a phenotypic test that may or may not be accurate.

Unfortunately, at this time there is not a better testing modality for sebaceous adenitis. While it is not generally a life threatening disease, it can be cosmetically debilitating for some dogs, and weekly bathing and skin care may be overwhelming for some owners. Until there is a genetic test or more accurate phenotypic test available, the skin punch biopsy should be used as a tool for better breeding decisions, even if there are some inconsistencies inherent in the test itself. As has been mentioned in previous articles, the use of testing procedures should be included along with pedigree analysis to determine the best pairing of a dog and a bitch within a breeding program. While SA symptoms can be controlled with special care, it would be nice if there were a genetic test available such as a DNA specific test that would enable breeders to identify carriers as well as affected dogs, and perhaps even to avoid the possible misdiagnosed animals that may be unnecessarily removed from a breeding program based on a flawed diagnosis. It may take a longer period of time to develop a DNA specific test if SA is a disease with multiple genes involved in its expression. Perhaps poodle breeders will be fortunate, and the universities currently studying the disease will discover a more accurate testing modality in the near future.