

Sebaceous Adenitis in Dogs

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Abstract: Sebaceous adenitis is an inflammatory skin disease of unknown etiology that leads to a poor haircoat. It occurs mostly in young adult to middle-aged dogs. Clinical signs vary in severity and distribution among breeds and types of haircoat. Lesions include alopecia, scaling, and follicular casts that are typically distributed over the face, head, pinnae, and trunk. Diagnosis is based on histopathologic findings of sebaceous gland inflammation and loss. Treatment for sebaceous adenitis involves lifelong management with various topical therapies containing keratolytic/keratoplastic agents, emollients, and humectants in addition to oral therapies such as ω -3/ ω -6 fatty acids, cyclosporine, and/or retinoids.

Sebaceous adenitis is an idiopathic skin disease characterized by an inflammatory reaction that targets the sebaceous glands.¹ Destruction of the sebaceous glands leads to a lack of sebum production, severely compromising the skin's natural structure and function. The haircoat becomes dry and brittle, with the possible development of secondary bacterial and/or yeast overgrowth or infections. Although it is an uncommon disease, sebaceous adenitis should be included in the differential diagnosis for dogs presenting with alopecia, follicular casts, and seborrhea sicca, especially in breeds such as the standard poodle, Akita, Samoyed, vizsla, Havanese, and English springer spaniel.

Pathogenesis

Sebaceous glands are distributed throughout all haired skin and produce an oily secretion known as *sebum*. Sebum is responsible for forming a surface emulsion over the stratum corneum that retains the moisture and hydration of the epidermis.^{2,3} The haircoat becomes dry and brittle if sebaceous gland function becomes inadequate. Sebum acts as not only a physical barrier but also a chemical barrier, with fatty acid constituents (linoleic, myristic, oleic, and palmitic acids) that exhibit antimicrobial properties.² The alopecia seen in sebaceous adenitis is thought to be due to perifollicular fibrosis caused by decreased function of hair follicle stem cells rather than destruction of hair follicles.⁴

Sebaceous adenitis is most often a primary idiopathic disease process; however, it has been seen as a secondary change with other dermatopathies such as leishmaniasis, demodicosis, uveodermatologic syndromes, and food allergy.⁵ The etiology of primary sebaceous adenitis is unknown, but multiple hypotheses have

Dr. McKay discloses that she is currently participating in a clinical drug trial sponsored by Pfizer.

been proposed: (1) a genetically inherited and developmental defect leads to destruction of the sebaceous glands; (2) an abnormality in lipid metabolism affects keratinization and sebum production; (3) a keratinization defect obstructs the sebaceous ducts; or (4) an immune-mediated or autoimmune disease is directed against the sebaceous glands.^{1,6} Phenotypic evaluation of cells present in sebaceous adenitis has shown marked infiltration of dendritic antigen-presenting cells and T cells, suggesting an immune-mediated pathogenesis for this disease.⁷ Sebaceous adenitis does not respond to corticosteroids,^{1,6} which may reflect a disease process different from that of other immune-mediated diseases. However, a cell-mediated immunopathogenesis is further supported by the disease's successful management with cyclosporine, an immunomodulatory drug.⁸⁻¹² Due to the overrepresentation of certain breeds, a genetic predisposition is also likely. In standard poodles and Akitas, the disease appears to be inherited as an autosomal recessive trait.^{4,13}

Signalment

Sebaceous adenitis occurs most commonly in dogs; however, it has also been reported in cats, rabbits, a horse, and humans.^{12,14-17} The disease tends to appear in young adult to middle-aged dogs and does not seem to have a sex predilection.^{1,3} Sebaceous adenitis has been reported in more than 50 different breeds of dogs, although breed predilections exist for standard poodles, Akitas, Samoyeds, English springer spaniels, and vizslas.^{3,18} Recently, sebaceous adenitis has also been recognized as a common dermatologic disease in Havanese dogs, although pedigree analysis is still needed to define a possible mode of inheritance.¹⁹



Figure 1. Follicular casts. (A) Note the accumulation of keratin and follicular material adherent to the hair shafts. (B) Silver-white follicular casts on the pinna of a Havanese.

Clinical Signs and Diagnosis

In general, the most common clinical signs among all breeds include follicular (keratosebacous) casts or fronds (FIGURE 1) with adherent scaling and bilaterally symmetric hair loss over the dorsal trunk, face, pinnae, and temporal region²⁰ (FIGURE 2). A “rat tail” can also appear with marked alopecia and adherent scale.¹

The clinical presentation of sebaceous adenitis depends in part on the breed of dog (BOX 1).^{1,6} In long-coated breeds such as standard poodles, Akitas, and Samoyeds, a symmetric partial alopecia is noted with excess scaling (may appear silver-white) and follicular casts affecting the dorsal muzzle, top of the head, pinnae, dorsal cervical region, tail, and trunk.²¹ The hair of standard poodles may become straighter, losing its curl, while Akitas may show more signs of complete alopecia/thinning haircoat and recurrent secondary pyoderma.^{6,21} Bacterial folliculitis can manifest as pustules, papules, or nodules. Some variation may occur; pyoderma has been noted to differ significantly between breeds.¹⁸

A second form of the disease involves short-coated breeds such as the vizsla. The disease usually presents as annular to coalescing areas of moth-eaten alopecia along with adherent scale affecting the ears, head, and trunk^{1,20,21} (FIGURE 3). Clinical signs may initially present as otitis externa secondary to buildup of scale in the ear



Figure 2. Bilaterally symmetric alopecia. (A) On the bridge of the nose. (B) Along the dorsum.

canal.¹³ In addition, a recent report described initial presentations of sebaceous adenitis as otitis externa with subsequent erosive to ulcerative pinnal lesions in three vizslas.²² Typically, sebaceous adenitis is not a pruritic disease unless concurrent pyoderma is present; however, pruritus associated with this disease has been reported in the absence of bacterial infection.^{18,23}

The differential diagnosis for sebaceous adenitis includes demodicosis, dermatophytosis, bacterial folliculitis, vitamin A-responsive dermatosis, ichthyosis, zinc-responsive dermatosis, endocrinopathy, and pemphigus foliaceus. It is essential to first assess for the common causes of folliculitis by obtaining surface skin samples for cytology to look for pyoderma, deep skin scrapings to rule out demodicosis, and dermatophyte culture to eliminate dermatophytosis. As secondary pyoderma is a common sequela, positive skin cytology results do not eliminate sebaceous adenitis as a differential. If follicular casts and alopecia are present, especially with a history of

Box 1. Clinical Signs of Sebacous Adenitis^{1,6,20,21}

Common clinical signs shared by most breeds

- Follicular (keratosebaceous) casts or fronds
- Scaling
- Alopecia and/or poor haircoat
- Pruritus, especially if concurrent bacterial or yeast overgrowth or pyoderma is present
- Papules, pustules, or epidermal collarettes if concurrent pyoderma is present

Common signs in long-coated breeds (e.g., standard poodle, Akita, Samoyed)

- Dorsal muzzle, top of head, dorsal cervical region, pinnae, tail, and trunk affected
- Symmetric partial alopecia
- Excess scaling and follicular casts
- Hairs may be brittle and dull; in white-coated dogs, they may be brown to red
- Matted hair
- Straighter hair, loss of curls in standard poodles
- Akitas may show more signs of complete alopecia and thinning haircoat

Common signs in short-coated breeds (e.g., vizsla)

- Ears, head, and trunk affected
- Annular to coalescing areas of moth-eaten alopecia
- Adherent scale
- Otitis externa

previous incomplete response to antibiotics, a skin biopsy should be the next diagnostic step.

Sebacous adenitis may be suspected based on clinical signs, breed, and history, but definitive diagnosis is made by histopathologic examination. Biopsy findings vary with chronicity of lesions. Acute lesions show granulomatous to pyogranulomatous inflammatory reactions targeting the sebaceous glands, and more chronic lesions may reveal a complete absence of sebaceous glands with focal perifollicular fibrosis.^{3,20} Hyperkeratosis may also be present in the epidermis and/or hair follicles. It is important to select multiple (three to four) sites for biopsy sampling, including alopecic lesions indicative of more chronic disease as well as early lesions characterized by adherent scale or follicular casting.²⁰

Treatment

Treatment for sebacous adenitis is twofold, involving both topical and systemic therapy. The goals of treatment are not only to remove excess scale and crusts while improving coat quality but also to reduce sebaceous gland inflammation and destruction.



Figure 3. Annular to coalescing areas of alopecia affecting the trunk and extremities.

Topical Therapy

The topical treatment regimen starts with a four-step process that, although labor intensive, provides excellent results:

Step one: Remove excessive scale, crusts, and follicular casts by bathing the dog in a keratolytic or keratoplastic shampoo.^a We recommend using shampoos with ingredients that have synergistic properties, such as sulfur and salicylic acid.²⁴ In addition, a medicated shampoo^b combining the keratolytic properties of sulfur with the follicular-flushing activity of benzoyl peroxide is effective for cases with secondary bacterial folliculitis.⁶ Shampoos should be allowed to sit for 10 minutes before rinsing completely.

Step two: Apply an oil treatment consisting of a 50:50 mixture of water and either a bath oil^c or generic baby oil to replace the oily barrier of the stratum corneum.^{3,25} This should be thoroughly rubbed into the entire haircoat and allowed to soak for 1 to 2 hours. It may be easiest to keep the dog in a kennel, crate, or small bathroom while allowing the oil treatment to soak.

Step three: Remove all the unabsorbed oil with a liquid dish-washing detergent.^d

Step four: Apply a moisturizing conditioner^e or humectant rinse/spray.^{f,25}

This regimen should be followed once a week for 4 to 6 weeks, then every 2 to 4 weeks as needed. If the haircoat shows improvement after 1 to 2 months, the oil soaks can be replaced by topical spot-on treatments containing essential oils and ω -3/ ω -6 fatty acids^g or 1% phytosphingosine.^h Essential oil spot-ons should be applied to the surface of the skin once a week for 8 weeks, then

^aExamples: Sebolut, Virbac, Fort Worth, TX; Keratolux, Virbac; MPA Seba-Hex, Vétquinol, Fort Worth, TX

^bExamples: DermaBenSs, Dechra Veterinary Products, Overland Park, KS; Oxiderm, Sogeval, Oldsmar, FL

^cAlpha Keri bath oil, Novartis Consumer Health, Parsippany, NJ

^dExamples: Dawn dish soap, Procter & Gamble, Cincinnati, OH; Palmolive dish soap, Colgate-Palmolive, New York, NY

^eExamples: Hydra Pearls, Vétquinol; EPI-SOOTH cream rinse and conditioner, Virbac

^fHumilac spray, Virbac

^gDermoscent Essential 6 spot-on, LDCA, Castres, France

^hDouxo Seborrhea spot-on, Sogeval, Coppell, TX



Figure 4. Effects of cyclosporine therapy in a shih tzu. (A) Before treatment. Note the “rat tail” and generalized thinning of the haircoat. (B) After 30 days of treatment. Good regrowth of the haircoat is evident.

once every 2 weeks for maintenance. Unlike the generalized oil soaks, these spot-on treatments should not be rinsed off. Volumes per dose (pipette) are 0.6 mL (<10 kg body weight), 1.2 mL (10 to 20 kg), and 2.4 mL (20 to 40 kg). Phytosphingosine spot-on treatments can be applied topically in two or three places in the shoulder and lumbar area twice a week until lesions resolve: 1 pipette (<20 kg body weight), two pipettes (21 to 45 kg), or three pipettes (>45 kg). These can also be applied every 1 to 2 weeks as maintenance therapy and could be ideal for more localized cases.

Spray-on therapy can be continued between baths, either with topical propylene glycol (mixture of 50:50 or 75:25 of propylene glycol and water)²⁵ or 0.2% phytosphingosine spray if the skin and haircoat remain dry between baths.¹ Propylene glycol acts as a hygroscopic agent, penetrating the stratum corneum and increasing its water content.²⁴ In addition, we typically use dietary supplements containing essential fatty acids, including ω -3 and

¹Douxo Seborrhea Microemulsion spray, Sogeval, Coppell, TX

ω -6 fatty acids, for potential antiinflammatory properties, barrier protection, and effects on lipid metabolism.^{21,24,25} Corticosteroids at either antiinflammatory or immunosuppressive doses do not seem to add any benefit to the management of sebaceous adenitis.^{1,6,21}

During this treatment regimen, clients who use a spot-on parasiticide should follow the labeled instructions for the specific product regarding how long bathing should be postponed after product application, particularly when the patient is being bathed once a week. Once the patient responds to treatment for sebaceous adenitis, the use of topical parasiticides should not pose any problems.

Systemic Therapy

In addition to topical therapy, many clinicians use oral cyclosporine³ as the systemic treatment of choice for sebaceous adenitis (FIGURE 4). Cyclosporine suppresses IL-2 transcription, thereby inhibiting the induction and proliferation of cytotoxic T cells and acting as an immunomodulatory agent.²⁶ In dogs, it has been shown to induce sebaceous gland regrowth in up to 40% of hair follicles after 12 months of treatment, in addition to significantly decreasing the severity of inflammation and mean clinical scores (extent of alopecia and follicular casts).⁸ Cyclosporine has also been shown to be a potent inducer of anagen or hair growth phase in mice, which, in theory, could inhibit the atrophy of hair follicles seen in long-standing cases of sebaceous adenitis.^{8,27} Adverse effects of cyclosporine include vomiting, diarrhea, gingival hyperplasia, hirsutism, and, rarely, papillomatosis, atypical bacterial or fungal infections, and psoriasiform dermatitis.^{28,29} Cyclosporine is used initially at a dose of 5 mg/kg once daily until clinical signs resolve and then gradually tapered.^{8,10} The tapering schedule can vary, but it is preferable to reduce the number of doses given per week, keeping the same total daily dose of 5 mg/kg (BOX 2). The need for measuring canine serum levels of cyclosporine is debatable because there are no studies evaluating trough levels of cyclosporine and efficacy for treatment of sebaceous adenitis. Amelioration in clinical signs should be seen within 4 to 6 weeks with aggressive topical therapy and oral cyclosporine, although significant clinical improvement may not be apparent until 4 months into treatment, depending on coat length.^{8,10} Although remission may be seen in rare cases, clients should be made aware that most dogs that respond to cyclosporine require lifelong administration.⁸

Combination Therapy

A multicenter, placebo-controlled clinical trial¹⁰ compared cyclosporine and topical therapy as sole therapies with their use in combination and evaluated efficacy based on alopecia, scaling, and histopathologic changes. Topical therapy consisted of salicylic acid/sulfur or ethyl lactate baths with baby oil soaks and 70% propylene glycol in water sprays. All dogs in all three treatment groups showed reduced alopecia and scaling, with a range of 57% to 81% improvement in alopecia after 4 months of treatment. The effect on sebaceous adenitis was similar between cyclosporine and topical treatment as sole therapies. However, when cyclosporine

¹⁰Atopica, Novartis Animal Health US, Greensboro, NC

and topical therapy were used together, alopecia and scaling scores were lower, suggesting that the combination of these two treatment modalities may have a synergistic benefit. Scaling improved up to 70% after 4 months with this combination compared with the 29% to 48% seen in the other two treatment groups.¹⁰ For this reason, we recommend the use of cyclosporine and topical therapy with a keratolytic shampoo, oil soaks, and moisturizing conditioners as our first-line therapy for treatment of sebaceous adenitis. We also add fatty acids and oil spot-on treatments as part of our therapy regimen.

Clients may have financial concerns or be unable to carry out the labor and time commitment of intense topical therapy. For mildly affected dogs, the use of antiseborrheic shampoos,⁶ oral fatty acid supplementation, and sprays (i.e., phytosphingosine or propylene glycol) is recommended as initial therapy. If frequent topical therapy is not possible, benefit can still be seen using oral cyclosporine¹⁰ and fatty acid supplementation alone, although this may not produce optimal results. In more chronic and severe cases, combination protocols using topical and systemic therapy are needed.

Alternative/Adjunctive Therapy

In cases that do not respond to topical therapy and oral cyclosporine, retinoids (vitamin A [retinol], isotretinoin,^k or acitretin^l) are also an option (**BOX 2**). In general, retinoids have antiinflammatory activity and influence cellular proliferation and differentiation, thereby playing a role in normalizing the keratinization process.^{24,30} Vitamin A appears to be a safe form of adjunctive therapy,²³ with one report³¹ showing 80% to 90% improvement after 3 months of therapy in certain breeds. For refractory cases of sebaceous adenitis, synthetic retinoids such as isotretinoin and acitretin can be effective. A retrospective study evaluating the use of synthetic retinoids in dogs with sebaceous adenitis showed more than 50% improvement in scaling and alopecia in 60% of dogs.⁵ Adverse effects of retinoids include keratoconjunctivitis sicca (KCS), teratogenicity, increased serum triglyceride concentrations, vomiting, diarrhea, and hepatotoxicity.^{5,30} For these reasons, dogs receiving treatment with retinoids should be monitored with Schirmer tear tests along with serum biochemical profiles, and retinoids should not be given to pregnant animals. Expense and restricted availability of isotretinoin are other concerns with the use of retinoids.

Although there have been no formal studies evaluating the efficacy of tetracycline and niacinamide for the management of sebaceous adenitis, anecdotal reports have described improvement in clinical signs using this combination protocol^{3,32} (**BOX 2**). Possible adverse effects of tetracycline and niacinamide include vomiting, diarrhea, anorexia, and lethargy. Rarely, liver toxicity is seen with niacinamide³³; we have also seen liver toxicity with tetracyclines.

Concurrent bacterial and/or yeast overgrowth or superficial pyoderma needs to be treated with an appropriate topical and/or antimicrobial therapy for at least 30 days, or 7 days beyond resolution

^kExamples: Amnesteem, Bertek Pharmaceuticals, Morgantown, NY; Accutane, Roche Pharmaceuticals, South San Francisco, CA; Claravis, Barr Laboratories, Pomona, NY

^lSoriatane, Stiefel, Research Triangle Park, NC

Box 2. Systemic Therapy for Sebaceous Adenitis

Cyclosporine^{5,7}

Start at 5 mg/kg once daily and then gradually taper to the lowest effective frequency of dosing. For example, once clinical signs have resolved, administer cyclosporine only 5 days a week for 1 month, then taper to every other day for an additional month. Continue to taper the cyclosporine to the lowest number of doses per week until the minimum frequency of dosing that keeps disease in remission is identified.

ω-3 and ω-6 fatty acids^{21,24,29}

180 mg of eicosapentaenoic acid (EPA) per 10 lb body weight per day

Antibiotics and/or antifungals

As needed for concurrent bacterial or yeast overgrowth or superficial pyoderma

Retinoids

- Vitamin A^{3,31}: 8,000 to 10,000 IU twice a day initially; can increase to 20,000 to 30,000 IU twice a day.
- Isotretinoin or acitretin^{3,5}: Start at a dose of 1 mg/kg PO q12–24h. If positive results are seen within 6 weeks, the frequency of administration can be decreased to 0.5–1 mg/kg q48h for maintenance.

Tetracycline and niacinamide³

- Dogs weighing <25 kg: 250 mg of each q8h
- Dogs weighing >25 kg: 500 mg of each q8h

of clinical signs and/or negative cytology results. We recommend treating uncomplicated superficial pyoderma with cephalexin, cefpodoxime, cefovecin, or amoxicillin/clavulanic acid. Concurrent use of sulfa drugs and retinoids should be avoided, as this combination can lead to increased risk for secondary KCS. Dogs receiving antibiotics with persistent bacteria present on cytology or clinical signs consistent with superficial pyoderma (e.g., epidermal collarettes, pustules, papules) require sample submission for culture and sensitivity testing. *Malassezia* overgrowth may require systemic administration of azole therapy (ketoconazole, fluconazole, or itraconazole) at standard doses for periods of 2 to 4 weeks.

Prognosis

Owners of dogs diagnosed with sebaceous adenitis need to be aware that this is a disease that typically requires lifelong management and may become progressive. Some dogs may undergo spontaneous remission or worsening with or without treatment.⁶ In addition, any secondary bacterial infections must be managed because they can contribute to worsening alopecia and pruritus. Some breeds, such as the Akita, may suffer from systemic illness or malaise, but this is not a common finding.^{13,20}

Response to treatment may vary depending on when diagnosis is made, as advanced disease is associated with diffuse destruction of sebaceous glands and perifollicular fibrosis (scarring).⁶ It is therefore important to keep sebaceous adenitis as a diagnostic

differential for alopecic and scaling/crusting dermatoses, as initiating treatment early in the disease process may improve the prognosis for long-term management.³⁴ Previous studies have also suggested that variations in the underlying causes of sebaceous adenitis between breeds may explain why clinical presentations and treatment success can differ in canine patients.^{5,23} It is recommended that affected dogs not be bred due to the potential hereditary nature of sebaceous adenitis.¹

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1. Sebaceous adenitis is commonly reported in

- a. Bernese mountain dogs.
- b. rottweilers.
- c. greyhounds.
- d. Samoyeds.

2. Which is a clinical sign of sebaceous adenitis?

- a. depigmentation
- b. "rat tail"
- c. pododermatitis
- d. vesicles

3. The differential diagnosis for sebaceous adenitis could include

- a. endocrinopathy.
- b. erythema multiforme.
- c. eosinophilic furunculosis.
- d. sterile nodular panniculitis.

4. _____ is not a proposed etiology for primary sebaceous adenitis.

- a. Inherited defect leading to sebaceous gland destruction
- b. Immune-mediated disease
- c. Abnormality in lipid metabolism
- d. Dermatophytosis

5. Treatment for sebaceous adenitis should not include

- a. cyclosporine.
- b. propylene glycol.
- c. corticosteroids.
- d. oil soaks.

6. Which is an adverse effect of retinoid therapy?

- a. gingival hyperplasia
- b. KCS
- c. hypercalcemia
- d. papillomatosis

7. Which is an effect of cyclosporine?

- a. suppression of IL-2 transcription
- b. keratolytic activity
- c. stimulation of fibroblasts
- d. antimicrobial and antifungal activity

8. A middle-aged dog with bilaterally symmetric alopecia and adherent scale initially presents with lesions on the dorsal cervical region and head. What would be an appropriate initial diagnostic test?

- a. biopsy
- b. cytology and deep skin scraping
- c. measurement of serum total thyroxine levels
- d. all of the above

9. Which statement is true regarding cyclosporine therapy for sebaceous adenitis?

- a. Administration of an appropriate dose of cyclosporine for 4 to 6 months often leads to remission of sebaceous adenitis with eventual discontinuation of therapy.
- b. Serum trough levels should be measured in all dogs in order to assess overall efficacy.
- c. Cyclosporine has a possible synergistic effect when used concurrently with topical therapy.
- d. If no improvement is seen within 10 to 14 days of initiating treatment, an alternative oral therapy should be administered.

10. Which statement regarding sebaceous adenitis is true?

- a. Sebaceous adenitis is proposed to be an autosomal recessive trait in certain breeds predisposed to the disease.
- b. Sebaceous adenitis commonly causes systemic illness.
- c. Retinoids and sulfa drugs have been shown to have a beneficial synergistic effect when used together for treatment of sebaceous adenitis.
- d. Sebaceous adenitis often presents as a focal alopecic lesion on the distal extremities.