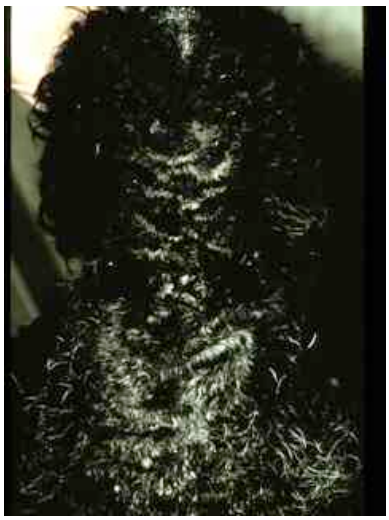
Sebaceous Adenitis

AETIOLOGY AND PATHOGENESIS

Sebaceous adenitis is an uncommon disease characterised by loss of sebaceous (oil) glands, scaling and hair loss. The aetiology and pathogenesis are unknown. In Standard Poodles the disease is hereditary and appears to be an autosomal recessive. There may also be a hereditary predisposition in Akitas and Hovawarts. Theories as to why the disease develops include an autoimmune response to sebaceous gland antigens or a primary structural defect of the sebaceous gland or its duct, which allows the leakage of sebum into the dermis where it provokes a foreign body reaction. During the early stages there is a mild inflammation about the hair follicles, which develops into a nodular granulomatous inflammatory reaction around the sebaceous glands. End stage follicles exhibit a lack of sebaceous glands and scaring about the hair follicle with keratin plugging of the follicular infundibula. It is not known if there is a causal relationship between the sebaceous gland changes and the abnormal follicular keratinisation, or whether these are simply coexisting features of a common inheritable process. Hair loss associated with sebaceous gland adenitis is thought to be a consequence of the parafollicular scaring interfering with growth cells of the follicle.

CLINICAL FEATURES

Sebaceous gland adenitis occurs in young-adult to middle-aged dogs, with no sex predilection. There is a breed predilection for Standard Poodles, Akitas, Hungarian Vizslas, and Samoyeds, but it is also seen in a wide variety of other breeds. The clinical appearance, distribution, and severity of lesions vary from breed to breed, and from animal to animal within a breed.



In Standard Poodles the first signs of clinical disease generally appear in young-adult to middle-aged animals with 90% of affected animals 1.5-5 years of age. Lesions first start on the top of the neck, head, back or ears. Initially, they appear as scaling and thinning of the hair, focal areas of hair loss and discolouration of the coat. There are usually prominent follicular casts - tightly adherent follicular debris around the base of the hair shaft. As the condition progresses, more areas of skin become involved with severe scaling, characterized by tightly adherent silver-white scales incorporating small tufts of matted hair. Hair loss becomes more severe as the condition advances. Secondary bacterial folliculitis commonly results in inflammation and pruritus. Samoyeds have

lesions similar to Standard Poodles, except that the scale tends to build up into plaque-like lesions.

Akitas exhibit similar clinical signs to Standard Poodles, but often have a more extensive hair loss, seborrhoea, and superficial bacterial folliculitis or deep bacterial folliculitis and furunculosis. They may also manifest systemic signs of malaise, fever and weight loss, especially if there is a severe secondary infection.

Hungarian Vizslas and other short-coated breeds have clinical signs that are characterised by multifocal annular and serpiginous areas of alopecia and fine white scaling that occur progressively over the head, ears, and trunk. The scaling may be the most prominent sign in Springer Spaniels.

DIFFERENTIAL DIAGNOSES

- Vitamin A responsive dermatitis
- Primary keratinisation defects
- Leishmaniasis
- Ringworm
- Demodectic mange
- Superficial bacterial infection
- Zinc responsive dermatosis
- Hypothyroidism
- Colour dilute alopecia
- Follicular dysplasia
- Pemphigus foliaceus
- Epitheliotropic lymphoma

DIAGNOSTIC TESTS

Biopsy of the skin reveals a multifocal inflammatory infiltrate of histiocytes, lymphocytes, neutrophils, and plasma cells around the sebaceous glands and other adnexal structures in early disease. Advanced cases have moderate acanthosis, hyperkeratosis, follicular hyperkeratosis and an absence of sebaceous glands. Multiple biopsies are frequently necessary to observe the diagnostic pathology.

MANAGEMENT

The prognosis is fair to guarded as therapy is palliative and response to various medications is variable. Early or mild cases may be helped by shampooing weekly or twice a week with a shampoo containing phytosphingosine, or a spray containing the same ingredient. Omega-3 and omega-6 essential fatty acid dietary supplements have been advocated. More severe or advanced cases may be helped by spraying or rinsing daily or every 2-3 days with a mixture of 50-75% propylene glycol in water. This helps soften and loosen scale before bathing.

The synthetic vitamine A compounds can result in improvement in some cases. These drugs are generally well tolerated but adverse effects can include decreased tear production, soft stools, and liver changes. They also can cause birth defects and should not be used in breeding animals nor handled by women of child bearing age unless on oral contraception. Hungarian Vizslas and possibly Springer Spaniels have a better response to vitamine A derivatives than other breeds. These compounds are expensive, and vitamin A may be an effective and cheaper alternative in some dogs

Atopica has been reported to help some animals that have not been responsive to the vitamine A derivatives.

As sebaceous adenitis in Standard Poodles is a genetic disease, its prevalence can be decreased by identifying and not breeding animals who are carriers or who are affected with the disease. To assist owners and breeders in identification of both normal and affected animals, the Sebaceous Adenitis Registry for Standard Poodles has been established. Current information about this can be obtained by contacting the Institute for Genetic Disease Control, P.O. Box 222, Davis, CA 95617, USA.

KEY POINTS

- Diagnosis is only made by histopathological examination of multiple (at least three, preferably five) biopsy samples.
- Management involves considerable effort, and control rather than cure