

“Sebaceous Adenitis” – a mysterious skin disease

Overview

Sebaceous adenitis (SA) is an uncommon inflammatory disease centred on the destruction of the sebaceous glands. The disease has been reported in many different dog breeds and also in mixed breeds. Nevertheless, a predisposition of Akita, Hovawart, Vizsla and Samojede is known.

The following part explains the actual status of scientific investigations/knowledge.

As already mentioned, the sebaceous glands have a central meaning in SA. The sebaceous glands (Glandulae sebaceae) are localized superficially in the hairy skin of dogs. They have an execution way to the skin surface, the epidermis. Via this connection (duct) the glands empty the sebum (2).

The oily sebum can be found on the surface of the skin and hair. This emulsion ordinarily serves not only for regularization of the humidity of the skin, but trains a physically chemical barrier against potential pathogens. Furthermore the sebum consists of inorganic salts and proteins, as well as interferons and immunoglobulines. This comprehensive protection mechanism guarantee against external causes.

Additionally, this function is supported by many released fatty acids.

It was found that the number and size of the sebaceous glands vary over the body regions of a dog. Especially large sebaceous glands are localized at the chin. In the neck and in the region of tale also a high number of sebaceous glands were observed.

However, in the toes and soles bale, nipples and in the nasal mirror there were no sebaceous glands found.

Today only one skin disease, coupled with attacked sebaceous glands, was described in veterinary medicine literature: "The sebaceous adenitis "

Sebaceous Adenitis

The Sebaceous Adenitis is a primary, selective inflammation reaction against the sebaceous glands. Although the disease has been reported in more than 50 other breeds, cats and rabbits (1,2,3). This illness can lead to the entire loss of the glands. The loss of the secretion does not seem to impair the hair growth. In more chronic cases the hair coat may become thin. The remaining hairs tend to have an adhered cast of follicular debris around the root.

As microscopic investigations (histological examinations) of skin biopsies pointed out, the inflammation character of SA goes through several different stages. Only after complete loss of the sebaceous glands the inflammation intensity decreases. Clinically the skin seems dry and scaly combined with many hairless places.

In general, the coat is dull, dry and fragile. In addition the smell of the dogs' changes: They smell like "three days carried socks".

As already mentioned, different inflammation degrees of the skin can be ascertained by microscopic investigations. At the beginning of the disease, histopathologic findings demonstrate different cell-types of the immune system in the sebaceous glands. The cells were described as perifollicular granulomas (aggregations of many cells). The aggregations consist of defensive cells, macrophages, lymphocytes, plasma cells and neutrophile granulocytes.

The hair follicles themselves seem to be not involved in the process. In the final stage of the disease all above mentioned immune cells have destroyed the sebaceous glands and microscopically no more gland-structure was recognizable (1). Young adult to middle-aged dogs are most commonly affected and no sex predisposition has been noted.

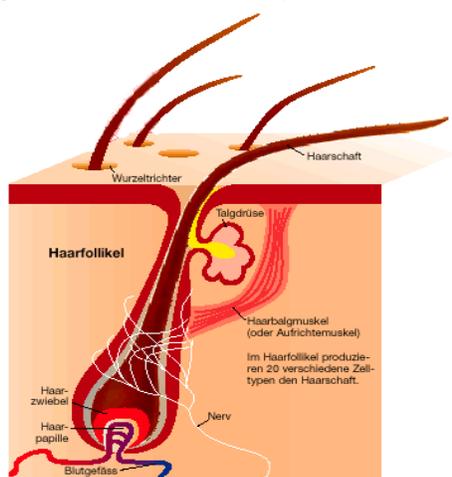


Fig: 1 Anatomy of the sebaceous gland

The first signs of the disease may be subtle and appear at the head, the ears and in the trunk. The hair seems thinner, almost "similarly to moth damage". In some cases the coat-colour changes.

SA is normally nonpruritic unless there is a secondary staphylococcal skin infection and even furunculosis may develop.



Fig.: 2 Clump of hairs (2)

The reason why sebaceous glands are attacked, is actually unknown. **In any case, no viruses, bacteria or other pathogens are observed as a primary cause.**

As shown in poodles **SA follows an autosomal recessive inheritance *1**, a so called genodermatose *2 (3). Furthermore it is supposed that also stress situations can trigger SA. However, cases of glucocorticoid medication seems to be also relevant for SA.

In addition no significant relations can be derived between the duration of the disease and the degree of SA. It has turned out, that it is very difficult to find out in which stage of the disease a SA-dog actually is. There are dogs with a nearly complete destruction of all glands after a timespan of 2 months. On contrary, individuals were monitored with a destruction of the glands for many years, similar to a creeping process. The disease has a very individual character (3). In some cases all cells of a gland were destroyed, and regeneration became impossible. But in advanced SA there are no gland-cell remnants.

From this point of view the scope of SA-research is actually focused on mechanisms triggering the destruction of the cells by the immune system and what kind of genetic reason(s) plays a key role.

Research

For the etiology of the disease microscopic investigations showed cells or other components of the immune system participate massively in the destruction process. However, no bacteria, viruses or other pathogens are proven as a cause of the disease, but the autoimmune reactions run against substances/cells belonging to body of the dog.

Interesting in this context was that, immunosuppressive drugs (Cyclosporin= Glucocorticoids *3) switched off the autoimmune attack and the disease delayed. On contrary when the drug was set down, the SA-dog falls back and the disease continue (1).

What causes exactly the false control of the immune system?

Until now the research has shown that **SA positive dogs have changes in the blood** which we cannot assign. In the blood many components of the immune system are transported. Finally these components can be found again later in the sebaceous glands, so the question is:

1. What sort of connections exists between the immune system / blood and the sebaceous glands?
2. What sort of genetic background triggers this process?

At the moment both questions were investigated by different methodical attempts. The organisation of immune system is very complicated and a huge number of interactions run off, so the search for causes is multi-layered. But to find the key and

the connections to the genetically triggered reasons, lots of investigations are necessary.

Without support of engaged breeders and Akita friends the progress of the SA-research would not be conceivable and we thank to all who help us.

PD Dr. Ina Pfeiffer

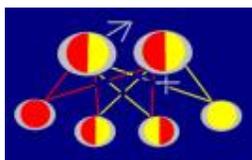
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Literature:

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- (3) Reichler IM, Hauser B, Schiller I, Dunstan RW, Credille KM, Binder H, Glaus T, Arnold S.:”
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Explanations:

***1:** Autosomal recessive Inheritance:



An abnormal gene on one of the chromosomes from each parent is required to cause the disease. Dogs with only one abnormal gene in the gene pair are called carriers, but since the gene is recessive they do not exhibit the disease.

In other words, the normal gene of the pair can supply the function of the gene so that the abnormal gene is described as acting in a recessive manner. BOTH parents must be carriers in order for a child to have symptoms of the disease; a child who inherits the gene from one parent will be a carrier.

***2 : Genodermatosis:**

A skin condition of genetic origin.

***3 : Glucocorticoids:**

Glucocorticoids are a class of steroid hormones characterised by an ability to bind with the cortisol receptor and trigger similar effects. Glucocorticoids are distinguished from mineralocorticoids and sex steroids by the specific receptors, target cells, and effects. Technically, the term corticosteroid refers to both glucocorticoids and mineralocorticoids, but is often used as a synonym for glucocorticoid. The name glucocorticoid derives from early observations that these hormones were involved in glucose metabolism. In the fasted state, cortisol stimulates several processes that collectively serve to increase and maintain normal concentrations of glucose in blood.

Glucocorticoids have potent anti-inflammatory and immunosuppressive properties