Sebaceous adenitis is a granulomatous or pyogranulomatous dermatitis with inflammation located around sebaceous glands. In chronic cases, these glands are replaced by fibrous connective tissue. Many breeds of dogs have been affected, but adult Standard Poodles, Samoyeds, Vizlas, and Akitas are apparently predisposed. Etiologic factors are unknown. Secondary pyoderma is often a complicating factor. Sebaceous adenitis should be considered in the differential diagnosis of dogs with seborrhea sicca that is unresponsive to medical therapy.

A 9-year-old, castrated male Samoyed dog was euthanized and presented to the Colorado State University Veterinary Teaching Hospital for necropsy. It had a 3-year history of a generalized dermatitis and blepharitis that was unresponsive to conventional medical therapy. Serologic and endocrine function tests were never performed.

The entire hair coat was thin, and the skin was rough, dry, and scaly. There was partial alopecia and crusting of the eyelids. The external ear canals were dirty and scaly. A postmortem examination of the abdomen, thorax, and brain revealed no other gross lesions. The tissues that were examined histologically were multiple areas of truncal skin, external auditory canal, upper and lower eyelids, nictitating membrane, thyroid, adrenal, hypophysis, and both eyes.

Sebaceous glands were absent in all areas of skin examined. Dorsal thoracic and dorsal lumbar skin had mild to moderate epidermal and follicular orthokeratotic hyperkeratosis and moderate infiltration around hair follicles by lymphocytes and plasma cells (Fig. 1). Over 150 sections from one sample of skin were cut and examined. No sebaceous glands were found in any section. Focal areas of epidermis were mildly acanthotic, varying from two to five cell layers thick. Small numbers of lymphocytes and plasma cells were scattered in the papillary dermis. Hair follicles were in catagen and anagen phases. Apocrine sweat glands were moderately dilated and were often surrounded by a lymphoplasmacytic infiltrate. Changes in ventral thoracic skin were similar but less pronounced.

Hair follicles in an external auditory canal were hyperkeratotic. Ceruminous glands and hair follicles were surrounded by lymphocytes and lesser numbers of macrophages, fibroblasts, and a few neutrophils. As in the skin, sebaceous glands were absent. Epithelium of an upper eyelid was acanthotic and had parakeratotic hyperkeratosis. A superficial crust consisted of keratin debris and neutrophils. Hair follicles were plugged with keratin and often contained neutrophils. Heavy infiltrations of lymphocytes, plasma cells, and macrophages surrounded hair follicles and the tubular glands of Moll. Tarsal glands, which are modified sebaceous glands, were absent. The nictitating membrane contained a normal amount of tubular lacrimal glands with small interstitial accumulations of plasma cells. There were dense accumulations of plasma cells in the subconjunctiva.

The zona fasciculata and zona reticularis of an adrenal gland were judged subjectively to be thickened in relation to the zona glomerulosa and the medulla. Examination of thyroid gland revealed no microscopic abnormalities. The eyes had no microscopic lesions.

In the adenohypophysis, multiple, large foci contained a lymphoplasmacytic infiltrate and scattered cells that were either chromophobic pituitary cells, which they closely resembled, or macrophages. These cells had vesicular, sometimes indented nuclei, and indistinct cytoplasm. Acidophilic and basophilic cells were greatly decreased or absent in the inflamed areas (Fig. 2). There was a pale, eosinophilic, loose, fibrillar matrix in affected foci. A Masson's trichrome-stained section failed to reveal an increased amount of collagen. The neurohypophysis was not affected.

Sebaceous glands are normally present in all regions of haired skin. The history, signment, distribution of skin lesions, and absence of sebaceous glands in this case are compatible with chronic or end-stage sebaceous adenitis of dogs. In contrast to those cases of sebaceous adenitis previously described in the literature, the dermatitis in this dog was not granulomatous. It is possible that the leukocytic infiltrate was modified as the sebaceous glands disappeared. It is also possible that this case represents a non-granulomatous variant of the disease syndrome.
Fig. 1. Truncal skin. There is epidermal and follicular hyperkeratosis. A mononuclear leukocytic infiltrate (arrow) is adjacent to a hair follicle. HE.

Fig. 2. Adenohypophysis. A mononuclear leukocytic infiltrate is in the parenchyma. Note the greatly decreased numbers of densely staining acidophils and basophils in the inflamed area. HE.

Microbiologic cultures taken from dogs with sebaceous adenitis often yield coagulate positive Staphylococcus, which is considered to be a secondary pathogen.\(^1\) Causative infectious agents have not been identified using aerobic, anaerobic or fungal cultures, or using electron microscopy.\(^1\) The disease syndrome is unresponsive to antibiotics, but will sometimes respond to immunosuppressive doses of corticosteroids if treated early in the course of the disease. This suggests an immune mediated etiology.\(^2\)

The lymphoplasmacytic adenohypophysitis described in this case is remarkable and resembles the inflammation around surviving adnexal structures of the skin. It is possible that an immune response is generated against a unique surface antigen that is shared by cells of the adenohypophysis and sebaceous cells. Such an inciting antigen could be a hormonal substance, secreted by the hypophysis, that binds to surface receptors on sebaceous cells. Alternatively, the dual inflammation may be coincidental.

Postmortem histologic examination of the hypophysis in suspected cases of sebaceous adenitis or chronic seborrhea sicca could determine whether or not inflammation of the adenohypophysis is a consistent finding in this disease.

References

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