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Cutaneous Manifestations of Canine Hyperadrenocorticism—Exogenous or Endogenous

The skin can exhibit a wide variety of lesions associated with excess steroids, regardless if the steroid source is endogenous or exogenous (iatrogenic). Since the overall effect of steroids is catabolic, one might expect to see atrophy, alopecia, weakness of skin and muscle, and other similar metabolic effects.

Some of the lesions illustrated below are more classically seen with steroid excess than others and most dogs with hyperadrenocorticism have at least 1 or 2 of the lesions. These lesions may be seen in any combination; however, it would be rare for a dog to have all or even many of these lesions.

Many of the following lesions are not unique to steroid administration or excess. Recognition can be quite problematic for veterinarians as can management. The veterinarian should use caution in evaluating unusual skin lesions; steroid therapy should not be administered if there is a chance that the lesions are caused by steroids. If in doubt, a biopsy is indicated and sample selection should represent the cutaneous lesion.

Noninflammatory Symmetrical Hair Loss

This is a rather nonspecific endocrine-associated finding. This dog has iatrogenic hyperadrenocorticism, which is manifested in the skin by symmetrical alopecia, thin skin, and a pot-bellied appearance. Another finding with hyperadrenocortisism is delay of hair growth after clipping.

Noninflammatory Hyperpigmentation

Pigmentary changes in the skin are usually symmetrical and accompany most endocrinopathies. In this poodle with endogenous hyperadrenocorticism, the increased pigmentation is more intense in areas of focal alopecia (A). In another patient the hyperpigmentation is more diffuse but also is associated with hair loss (B).

Noninflammatory Symmetrical Hair Loss & Calcinosis Cutis

This dachshund, a predisposed breed, has endogenous hyperadrenocorticism. Among the cutaneous findings are a poor hair coat, thin skin with poor elasticity, and inflammatory calcinosis cutis over the lateral rump area (A). Marked alopecia of the tail (commonly called "rat tail") is seen in the patient as well (B). A skin scraping has been taken from the shoulder.

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Classic Thin & Hypotonic Skin

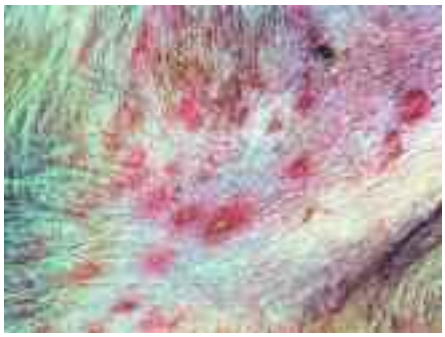
This is usually most apparent on the ventral abdomen and may be noted as tissue-paper–thin skin that lacks elasticity and allows the subcutaneous vessels to show through clearly. As skin atrophy becomes more severe, fine folds of skin or stria (stretch marks) may be present. Phlebectasias or tiny red, slightly raised spots representing abnormal dilatation, extension, or reduplication of small veins can be seen as a marker of canine hyperadrenocorticism.

Comedones

These keratin-filled follicles (found mostly around the nipples) may be black or white and, in some cases, are also associated with mineralization. Always consider *Demodex* if comedones are present.

Demodicosis

Dogs with excessive steroids, especially those with iatrogenic hyperadrenocorticism, may present primarily with adult-onset generalized demodicosis with or without pyoderma. The appearance of dark comedones or presumed hyperpigmentation warrants deep skin scrapings to identify *Demodex*. Secondary pyoderma, generalized demodicosis, and hyperadrenocorticism in a single presentation will require treatment in a step-by-step process.



Pyoderma

The only cutaneous finding in some dogs with hyperadrenocorticism may be superficial recurrent or partially responsive pyoderma. Pyoderma, especially deep pyoderma, is often a complication of calcinosis cutis.

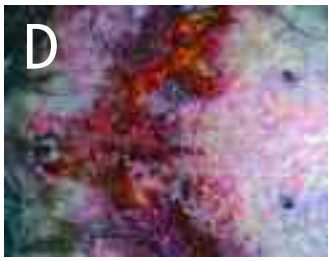
Calcinosis Cutis

This condition is unique to dogs and occurs when mineralization occurs in the dermis and along the follicular epithelium. It can occur both with endogenous and iatrogenic hyperadrenocorticism and the exact mechanism is not well understood. Calcinosis cutis is frequently located in intertriginous areas, such as the axilla and groin, but can also be found anywhere on the ventral abdomen or dorsum. Lesions over the dorsal neck are fairly common in severe cases. Mineralization can also develop in areas of previous trauma.

Early calcinosis may be noninflammatory and appear as pearlescent whiteness in a macular pattern superficially in the skin (**A**). Even at this stage, most affected areas of skin feel somewhat firm. Early lesions may also include those that appear pustular, but are firm and contain grit-like material, a product of dystrophic calcium deposition (**B**). Osseous metaplasia has rarely been described.

As the body reacts to the mineral material, a foreign body reaction with marked inflammation develops (**C**). These inflamed lesions may develop secondary bacterial for fungal infections (**D**) with exudation, which usually worsens the clinical sign of pruritus.

Diagnosis is made by biopsy, and treatment is best managed by determining the source of the steroids and decreasing or eliminating them. Systemic antibiotics should be given for pyoderma and generally should be used until the skin lesions have resolved. Topical treatment with DMSO gel has been advocated to help dissolve the mineral deposits locally. With treatment, inflammation begins to resolve and scaling may be noted (**E**).



Additional Signs

There are additional cutaneous signs of hyperadrenocorticism noted in the literature. For example, facial dermatosis has been described in 4 dogs as the only cutaneous manifestation.[1] Bruising is not uncommon and is especially noticeable when venipuncture is necessary. Nonhealing wounds are another sequela to hyperadrenocorticism. The diagnosis and treatment of hyperadrenocorticism is important to the well-being of the pet. References are available to assist with choosing the best test and treatment for the individual patient.

Acknowledgement

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