Feline paraneoplastic alopecia is a ventral alopecia in which the abdominal skin appears to glisten (but is not fragile), may have a secondary Malassezia infection, and has also been associated with dry, fissured foot pads. Necropsy usually reveals an exocrine pancreatic adenocarcinoma, often with metastases to the liver and elsewhere. One cat with a bile duct carcinoma has also been described. Temporary resolution of the skin disease has been reported in one cat which had the primary tumor removed; the lesions recurred following metastases of the tumor. Histology of the skin shows severe atrophy and miniaturization of the hair follicles.

Nodular dermatofibrosis syndrome in German shepherd dogs and occasionally other breeds associated with renal cystadenocarcinomas or cystadenomas has been reported. Histologic study of the nodules reveals dense collagen fibrosis. These nodules are most often found on the distal extremities. Diagnosis of renal lesions is best done by ultrasound. This should be repeated at six month intervals if the disease is suspected but the original ultrasound is normal. While the prognosis is serious, some dogs with benign renal cysts have survived for five years or more after diagnosis. Recent data in the German Shepherd dogs suggest that the syndrome may be caused by a mutation in a previously unidentified tumor suppressor gene.

Thymomas have been associated with an exfoliative dermatitis described in older (often ‘orange’) cats. The exact mechanism is unknown, but an erythema multiforme-type reaction has been proposed. If detected, the tumor’s removal will lead to resolution of signs. Recently, radiotherapy has been reported as a therapeutic option.

**Superficial necrolytic dermatitis**

Also known as SND, hepatocutaneous syndrome, epidermal metabolic necrosis, or diabetic dermatosis, this disease is being seen with increased frequency in dogs, and has also been reported in the cat and a red fox (*Vulpes vulpes*). The cutaneous lesions include crusting, erythema, exudation, and alopecia periorally and periconcularly, around the genitals, and the distal extremities, as well as hyperkeratosis and ulceration of the footpads. The skin disease may precede the onset of the signs of the internal disease. Histopathologic findings include superficial perivascular-to-lichenoid dermatitis, with marked diffuse parakeratotic hyperkeratosis and striking inter- and intracellular edema limited to the upper half of the epidermis (‘red, white and blue sign’). Diagnosis is usually made by clinical signs, confirmatory histopathology, and an ultrasound finding of both hyperechoic and hypoechoic areas in the liver (‘Swiss-cheese’ or ‘honey comb’ pattern).

Superficial necrolytic dermatitis resembles the glucagonoma syndrome (necrolytic migratory erythema) of humans, which is usually associated with hyperglucagonemia and a glucagon-secreting alpha-cell neoplasm of the pancreas. Hyperglucagonemia has also been documented in dogs with this syndrome; however, dogs tend to have hepatic parenchymal damage much more commonly than gluconomas. Dogs with SND have profoundly low levels of plasma amino acids.

Therapy is best effected with the infusion of amino acids (ex: Amnosyn®) given intravenously in a central vein, at an approximate rate of 60-80 mg/kg/24 hr. Osmalality and/or neurologic signs should be monitored (although problems are relatively uncommon). This is often performed on a daily basis for 2-3 days, and may need to be repeated on a q 3-6 week basis.

An alternative is the use of oral medications: ProCel® Powder (1 scoop/5kg q12h; Global Health Products www.globalhp.com 1-800-638-2879) or whey protein powders, scrambled eggs (? /day), elemental Zn (2 mg/kg/ day), and sAME (or similar liver protectants).

When the underlying disease can be treated (drug-induced hepatopathy, removal of glucagonoma) and secondary skin infections (bacterial and/or yeast) are treated, these dogs have usually responded well for variable lengths of time, sometimes for more than one year.

**Metastatic pulmonary carcinomas**

This problem has been reported in cats: the lesions occur on the distal extremities, especially the front feet, and look more like inflammatory pododermatitis than a neoplastic process. Their presence may be noted before pulmonary signs are noted. Rarely, the neoplasm may metastasize to other areas on the body, such as the abdominal skin. The neoplasm may be either a bronchogenic or squamous cell carcinoma. Paliative treatment to reduce the discomfort may be attempted (topical or systemic corticosteroids to reduce edema, etc).

**Endocrine disease**

Hypothyroidism, hyperadrenocorticism, and testicular neoplasia (particularly Sertoli cell tumor) may all cause cutaneous signs. Some of their presentations will be covered – *their diagnosis and treatment is covered elsewhere in this Convention*. 
**Hyperadrenocorticism**
Dermatologic signs noted in dogs with hyperadrenocorticism but not limited to that disease include:

1. Nonpruritic alopecia
2. Nonpruritic hyperpigmentation
3. Recurrent pyoderma
4. Recurrent dermatophytosis or demodicosis
5. ‘Seborrhea’ (excess scale, crust, etc)
6. Easily bruised skin

More specific clinical signs are:

1. Comedones
2. Calcinosis cutis
3. Thin skin
4. Perianal adenomas and hypertestosteronemia in females or neutered males

In one five year retrospective study, clinical features of hyperadrenocorticism were evaluated in 60 dogs that had cutaneous lesions as the first noticed clinical signs. Diagnosis was made by the ACTH stimulation test, the low dose dexamethasone suppression test, and the final diagnoses were 58 dogs with pituitary dependent hyperadrenocorticism, and two dogs with adrenal neoplasia. In retrospect, some of these dogs may have had non-classic adrenal disease (‘adrenal sex-hormone’ abnormalities), although only 5 of the 60 dogs (8%) were a breed typical of this syndrome (all were miniature poodles). Interestingly, two dogs were related (mother/son). Similar reports of related dogs have been reported.

While not referenced against the population at large, the most common breeds were poodles, dachshunds and Lhasa Apsos. Other breeds reported to be at risk are boxers and Boston terriers.

The most common cutaneous lesions were truncal alopecia (62%), pyoderma (55%) and hyperpigmentation (43%). Less common dermatologic manifestations were thin skin (14%), non-truncal (face, head) alopecia (13%), *Demodex canis* infestation (8%), and comedones (5%); only one had calcinosis cutis. Pruritus, seen in 15% of the dogs, was always associated with pyoderma, seborrhea, demodicosis, and/or calcinosis cutis.

**Hypothyroidism**
Dermatological features occur in at least 80 per cent of cases. The classical description is of bilaterally symmetrical, non-pruritic alopecia which spares the distal limbs, with varying degrees of hyperpigmentation and thickened, non-pitting skin (myxoedema). In fact, myxedema is uncommon, while the alopecia, with or without hyperpigmentation, may be focal, multifocal, regional or generalized and asymmetrical. The tail may lose most of its hair (‘rat-tail’), as may the dorsal muzzle. The haircoat may be easily epilated and may fail to regrow or regrow only slowly after clipping. In some breeds, particularly the Boxer and the Irish Setter, the hair may not be shed resulting in hypertrichosis (‘carpet-coat’). In the Golden Retriever and the Irish Setter, the ‘feathers’ on the legs may become a yellow color (‘blond frizzies’). Seborrhea is relatively common and may be dry, greasy or inflamed. Comedones may be present, particularly over the dorsum. Hyperkeratotic plaques may occur around the borders of the pinnae. Bacterial folliculitis occurs frequently. In the presence of seborrhea or pyoderma, hypothyroidism may be accompanied by pruritus.

**Sertoli cell tumor**

**Clinical signs**

- Nonpruritic alopecia
- Nonpruritic hyperpigmentation
- Enlarged nipples and/or mammary glands
- Palpable mass in testicle (NOT always present)
- Linear preputial dermatosis (may also be seen in any dog receiving estrogen treatment.)
- Occasionally, severe papular, pruritic rash
- Thrombocytopenia, anemia may be present (petechia)

**References**


