Introduction

Non inflammatory alopecia is a feline cutaneous reaction pattern and can be caused by a number of different diseases. As such it provides one of the true diagnostic challenges to the veterinary dermatologist. The term is used to describe an acquired syndrome of multifactorial aetiology that results in loss of hair over the perineum, proximal ventral and ventrolateral tail, hind limbs, ventrum, lateral abdomen and distal forelimbs and on rare occasions extending to the lateral thorax. The dorsum is not usually affected and the skin does not appear inflamed.

Feline non inflammatory alopecia may be self inflicted from licking, biting or pulling the hair as a result of pruritic dermatoses or conditions causing psychogenic disturbances or it may result from spontaneous hair loss due to either epilation of hair or hair shaft fracture. When presented with a case of non inflammatory alopecia in the cat, it is therefore critical to ascertain whether the cat is licking the hair out or whether the hair is falling out. Most, if not all, such cases are the result of pruritus or self-trauma due to an underlying problem, and are not related to sex steroids or other hormones.

Differential diagnosis for non inflammatory alopecia and self inflicted hair loss

Ectoparasites
Demodicosis
Cheyletiellosis
Otodectes cynotis

Infections
Dermatophytosis

Hypersensitivities
Adverse food reactions
Atopic dermatitis
Flea bite hypersensitivity
Drug eruption

Psychogenic
History

A history of pruritus in the cat may be difficult to elicit, as many cats will displacement groom rather than scratch. In many cases, the owners do not believe that the cat is pruritic because they have not seen any evidence of scratching. One of the challenges to the clinician is to persuade owners that alopecia can be self induced and not a spontaneous problem.

The following information should be obtained

- Breed
- Age of onset
- Distribution, progression and seasonality
- Lifestyle, environment and diet
- Previous illness
- The efficacy of prior therapy
- Evidence of contagion

Clinical examination

Hairs in both affected and non affected areas should be closely examined to determine whether the hairs have broken off (these feel stubbly to touch with normal resistance to epilation) or if they are easily epilated (removed easily to leave bald patches of skin). The skin should be examined to determine other lesions that might suggest pruritus (papules, erythema, excoriations).

Based on history and physical examination, it may be possible to determine if hair loss is due to pruritus or if it is spontaneous. If this is not possible, or in order to provide convincing evidence for the client, a trichogram should be performed.

Approach to self inflicted hair loss

Having established that hair loss is self inflicted, the clinician must then decide whether the cat is pruritic or psychogenic. It is imperative the clinician identify and eliminate all the potential causes of pruritus prior to making a diagnosis of psychogenic dermatitis. Bear in mind that in any individual cat the clinical picture may represent a combination of pruritic and behavioural aspects.

Step 1

A coat combing and superficial and deep skin scrapings to evaluate for ectoparasites (e.g fleas, flea faeces, Demodex, Cheyletiella, Otodectes species). If the tests are positive, the cat should be treated for the specific disease identified.

A Wood’s lamp examination should be performed. When present, fluorescent hairs should be plucked for fungal culture. If negative then a sample should be obtained with a toothbrush and submitted for fungal culture.
If the skin scrapings and Wood’s lamp examination are negative, then implement a flea therapeutic trial. In the cat we usually use either fipronil spray 6ml/kg applied topically q 7 days or oral nitenpyram 1mg/kg q 48 hrs for 30 days or both.

**Interpretation of the therapeutic trial**

A **good response** (>80% improvement) is consistent with the diagnosis of FBH. A long term maintenance flea control program is then instituted.

A **moderate response** (30-80% improvement) warrants continuing the therapeutic trial for a further 14 to 21 days and then reassessing the patient. If there is no further response (still 30-80% at day 42) there may be concurrent disease eg FBH and feline atopic dermatitis.

A **poor response** (< 30% improvement) suggests the diagnosis of FBH is unlikely and further investigation of other causes is warranted.

**Step 2**

If there is no change after four to six weeks and presuming the fungal culture is negative then I usually check the cat again for evidence of ectoparasites. I usually implement an acaricidal trial using either selamectin 6mg/kg applied topically q 14 days for six weeks of all affected and in contact animals. Selamectin is not licensed for this purpose. At the same time we usually begin an elimination diet trial to investigate the possibility of an adverse food reaction (AFR).

**Stage 3**

A diagnosis of an AFR relies on observing improvement of clinical signs when the cat is fed a novel protein diet followed by a recurrence of clinical signs when the original diet is reintroduced. The offending allergens then can be identified by a sequential challenge diet in which single ingredients (fish, beef, dairy, pork, chicken, lamb/mutton, eggs, wheat) are introduced to the elimination diet once a week to determine which individual allergen or allergens is incriminated. In our experience, most cats are only allergic to a single protein.

If there has been no response to the elimination diet then the cat should be evaluated for feline atopic dermatitis. A clinical diagnosis of atopy should be made on the basis of a compatible history in conjunction with ruling out an adverse food reaction and flea bite hypersensitivity dermatitis. Recommended treatments for feline atopy include allergen specific immunotherapy, allergen reduction, cyclosporin, glucocorticoids, antihistamines and fatty acids. Intradermal skin testing is performed to select allergens for an immunotherapy regime and to implement allergen avoidance measures. It is more technically demanding to perform in the cat than in the dog and requires referral to a veterinary dermatologist. If an owner elects not to pursue intradermal allergy
testing then commence symptomatic therapy. The medical management of the pruritic cat has been covered elsewhere.

If the testing is negative then two possible diagnoses remain; either the cat has psychogenic alopecia or an undiagnosed allergic skin disease caused by an environmental or dietary allergen. Unfortunately neither elimination diet trials or intradermal allergy testing are 100% sensitive for the diagnoses of allergic diseases.

Psychogenic alopecia has been covered in substantial detail in the previous lecture. In our dermatology referral practice, psychogenic alopecia is uncommon as a sole primary clinical entity in the cat. The possibility of both a pruritic component combined with a psychogenic component should be considered in some cases. We would estimate that 10% to 15% of cats with chronic non inflammatory alopecia in our referral dermatology practice have a psychogenic component reinforcing a primary pruritic skin disease. This may account for the partial success seen in some cases with symptomatic anti pruritic therapy

**Approach to Spontaneous Hair Loss**

If the history, physical examination and trichogram have indicated that the hair loss is spontaneous, the following approach should allow a definitive diagnosis to be achieved. It must be emphasised however that with the exception of dermatophytosis, the following skin conditions are much less common.

**Differential diagnosis for non inflammatory alopecia and spontaneous hair loss**

**Ectoparasites**
Demodicosis

**Infections**
Dermatophytosis

**Endocrine diseases**
Hyperthyroidism
Hypothyroidism
Hyperadrenocorticism
Diabetes mellitus

**Systemic disease**
Feline paraneoplastic alopecia
FeLV and FIV infection

**Defluxions**
Anagen defluxion
Telogen defluxion
Iatrogenic
Glucocorticoid or megoestrol acetate administration

Step 1

Check the history for administration of drugs (glucocorticoids or megoestrol acetate) or illnesses that could have triggered anagen or telogen defluxion. Feline anagen defluxion can be caused by antimitotic cancer or chemotherapy drugs, viral illness such as feline leukemia virus (FeLV), feline immunodeficiency virus (FIV) and feline infectious peritonitis (FIP) and metabolic disease such as diabetes mellitus. Telogen defluxion can be triggered by an abrupt stressful circumstance such as pyrexia, systemic illness, pregnancy and lactation or major surgery.

Cushing’s syndrome manifests as polyuria, polydipsia, polyphagia and abdominal enlargement. A syndrome involving polyphagia, polydipsia, polyuria associated with weight loss, hyperactivity, vomiting or diarrhoea in an older cat also suggests hyperthyroidism, which can sometimes produce alopecia associated with atrophic, hypotonic skin. Most cases of diabetes mellitus will present with polyuria, polydipsia, weight loss, polyphagia or inappetence. A change in general body condition and gastrointestinal signs are common in paraneoplastic alopecia relating to pancreatic or biliary neoplasia.

Superficial and deep skin scrapings should be performed to rule out demodicosis and a Wood’s lamp and fungal culture should be performed as previously described to rule out dermatophytosis. A blood sample should be collected for haematological and biochemical analysis, total T4 (thyroxine) concentrations and FeLV and FIV serology and a urine sample should be collected by cystocentesis. These tests may be supportive, or diagnostic of certain endocrinopathies.

Step 2

If all diagnostic tests are normal at this stage I usually proceed with a skin biopsy for histopathologic evaluation in conjunction with thoracic radiography and abdominal ultrasound.

A skin biopsy can be supportive of a diagnosis of telogen or anagen effluvium, hyperadrenocorticism or paraneoplastic alopecia. Abdominal ultrasound is useful for detecting enlarged adrenal glands and pancreatic or other visceral neoplasia.

Step 3

Specific treatment should be initiated if a specific diagnosis has been achieved with the preliminary testing. However it may be necessary to follow up screening tests with more specific endocrine function tests.
If hyperadrenocorticism is suspected then an adrenocorticotrophic hormone (ACTH) response test and/or dexamethasone suppression test is recommended. Adrenal function tests are not well established in the cat. Measurement of endogenous ACTH concentrations may be of benefit. Abdominal ultrasonography may provide evidence of unilateral or bilateral adrenal enlargement. Exogenous administration of progestagens or glucocorticoids should also be excluded.

In cats with suspect hyperthyroidism with normal or marginally elevated total T4 levels a T3 suppression test may be indicated. Radionuclide thyroid scanning is available in certain specialist referral centres.

**Step 4**

If all diagnostic tests are normal and the cat appears healthy apart from the alopecia the clinician has two options. The first is scientific neglect. The previous tests have ruled out any serious medical disorders requiring treatment and the client may prefer not to treat the cat and accept the alopecia as an aesthetic problem.

The second option is to consider the option of administering symptomatic therapy. A controversial concept associated with cats with normal thyroid function tests that have a decreased response to thyrotropin stimulation has led to the term “low thyroid reserve.” Thyroid supplementation with liothyronine has been advocated in these cats.

Alternative therapies include repositol injections of testosterone and diethylstilboestrol or progestagen therapy. There is no scientific rationale for using these drugs and the potential adverse effects are serious and in some cases irreversible. Most dermatologists strongly advocate these drugs be avoided.

**References**


