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Challenging Dermatology Presentations Mini Series

Session 2: The Pruritic Cat

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Introduction

The most common clinical presentation of feline skin disease is pruritus which has a variety of manifestations in the cat. The investigation and treatment of feline pruritus can be a major challenge. The diagnostic approach to feline pruritus does not differ substantially from that of the dog. However, the challenge with cats is firstly recognizing the diverse clinical presentations of feline pruritus and secondly, having made a diagnosis and formulated a treatment plan... actually implementing the treatment!

Pruritus and the feline reaction patterns

Pruritus in cats tends to be manifest in a limited number of ways, known as the feline reaction patterns. Compared to dogs, the distribution of pruritus and lesion type in cats is of lesser diagnostic significance. The feline cutaneous reaction patterns are:

1. **Miliary dermatitis**

2. **Symmetrical alopecia**

3. **Eosinophilic granuloma complex lesions**
    - The lesions of the eosinophilic granuloma complex may occur separately or in combination.

    - Eosinophilic plaques are well demarcated, alopecic, raised, glistening plaques with a moist, red surface. Usually located on the ventral abdomen, medial thighs or thorax. These lesions are very pruritic.

    - Eosinophilic (linear) granuloma are well defined papular or cord like, linear lesions usually occurring over the caudal hind limb but sometimes also seen over the thoracic wall, feet or oral cavity. These lesions are usually pruritic.

    - Indolent ulcers are well demarcated, raised, full thickness ulcerations that do not bleed situated unilaterally or bilaterally on the upper lips.

4. **Head and neck pruritus**

    - Frequently the most severe cutaneous reaction pattern and the most refractory to treatment.

Cats may present with one or more of these patterns at any one time.

The recognition of a reaction pattern does not constitute a diagnosis; it is only a manifestation of pruritus. Further investigation is indicated for cats that repeatedly present with one or more of these reaction patterns in order to try and establish the underlying cause of the skin lesions and institute specific therapy. A systematic approach to the diagnosis is required. The work up involves ruling out ectoparasitic disease (particularly flea allergy dermatitis), treatment of bacterial and/or yeast infection and investigation of the involvement of food and environmental allergens. In some cats a definitive diagnosis cannot be made and these cases are documented as idiopathic. Such work ups can be protracted and it is essential that the client understands from the outset the potentially lengthy and difficult nature of the investigation.

The approach to the pruritic cat

A systematic approach will maximise the chances of making a diagnosis and improve subsequent case management. The following steps should be undertaken:

History taking

The information gleaned from a detailed and, hopefully, reliable history is of paramount importance when drawing up the list of possible differential diagnoses.

The following areas should be covered during history taking.

- Is there evidence of systemic involvement?
- When did the skin disease start?
- Evidence of pruritus
- How did lesions first appear and how have they progressed?
- Seasonality?
- Evidence of contagion or zoonosis
- Diet
- Flea control used/ response to other treatments

Physical examination

A full physical and skin examination should be performed and lesions should be recorded.

Differential diagnosis

When drawing up a list of differential diagnoses with a view to further investigation, it is important to initially rule out / treat parasitic infestations before considering the infectious and allergic causes of pruritus.

- parasites
- infections
- allergies
- others

Diagnostic tests

The following diagnostic tests are indicated when presented with one of the feline reaction patterns and indeed in many other feline skin diseases. They are helpful to detect the presence of parasites, bacterial and yeast infections, and eosinophilic diseases.

Trichography

Trichography is the technique of microscopic examination of hair shafts. This is a useful test in cases of feline symmetrical alopecia where it is not clear that the cause of the alopecia is due to self-trauma. Hair tips taper to a fine point. Fractured hair shafts indicate self-trauma due to pruritus. Trichography can also be used to ascertain telogen to anagen ratios and may be useful in ectoparasitic and fungal diseases. Around 70% of hair follicles can be expected to be in the telogen phase in both normal cats and cats suffering from symmetrical alopecia.[1]

Tests for ectoparasites

Combings and brushings

Flea combing can be rewarding and certainly, if fleas are present in large numbers, they should be seen. However, if flea numbers are scarce, combing is an insensitive test. Cats tend to remove evidence of flea infestation by grooming.

Microscopic examination of coat brushings is a useful test for the diagnosis of surface and superficial parasites such as fleas, lice, harvest mites and *Cheyletiella* spp mites. Scale from the cat is collected onto paper and then transferred to acetate tape that is mounted onto a glass slide and examined under low power light microscopy.

Skin scrapings

Used for identification of superficial and deep parasitic mites such as *Cheyletiella, Sarcoptes* and *Demodex* spp. Avoid scraping areas that are excessively crusted or excoriated as this may lead to false negative results. Hair should be clipped. When scraping for *Demodex cati,* it helps to gently squeeze the skin between thumb and forefinger as this extrudes mites from the hair follicles. A small amount of liquid paraffin, or water if using potassium hydroxide, is applied to the area. A blunted No 10 scalpel blade is used to scrape material from the skin surface which is then mounted onto a glass slide in liquid paraffin or sodium hydroxide. When scraping for surface-living parasites such as *Cheyletiella* spp or harvest mites, make a superficial scrape of a larger area. Deeper skin scrapes resulting in capillary oozing should be made when looking for *Sarcoptes* or *Demodex* spp mites.

As these tests have a low sensitivity, trial therapy should be performed where ectoparasitism is one of the differentials but external parasites cannot be detected.

Cytology

Cytology is a fundamentally important technique in veterinary dermatology that can be employed quickly, easily, and inexpensively in a practice situation. Cytology frequently yields useful information on a case, and enables a more precise diagnosis to be made so that an accurate prognosis can be given. It will greatly enhance the chances of therapeutic success.

Techniques for taking and staining cytology samples and their interpretation will be discussed in a later lecture.

How do I know it's not parasitic?

Fleas

Flea bite hypersensitivity (FBH) is the most common feline skin disease on a worldwide basis (although perhaps not in northern climes) and can result in any of the feline cutaneous reaction patterns. The aetiopathogenesis in dogs is considered to be both type I and type IV hypersensitivity responses to flea salivary antigens and also possibly late phase reactions. Less is known about the pathogenesis of flea allergy dermatitis in cats although most flea allergic cats have positive intradermal test reactions to flea antigen and have flea allergen specific circulating IgE.[2,3]

The first stage in any case of feline pruritus is to make absolutely certain that fleas are not involved. Remember that overgrooming in cats with FBH effectively removes evidence of fleas or flea faeces in the hair coat. Flea allergic cats may be identified by the presence of immediate and/or delayed reactions to intradermal injection of flea extracts but false negative results occur. The use of the FcεR1 molecule detection system and the use of flea salivary antigens rather than whole flea extracts to detect the presence of circulating IgE antibodies may offer a more reliable diagnostic test in cats[2,4] However, a negative result does not rule out flea involvement and may result in further reluctance on the part of the client to institute a flea control program.

The response to a rigorous flea control programme is perhaps the only sure way of identifying flea allergic animals. Aggressive flea control is required for at least six weeks using an effective adulticide on all pets in combination with the use of an adulticide and insect growth regulator for environmental treatment (indoors and sometimes outdoors). A good knowledge of the flea life cycle is essential. Since allergic cats overgroom, they may remove products applied topically, resulting in apparent treatment failure. The use of a Buster collar for 24-48 hours following application of topical therapy should prevent this. There is no topical adulticide that will act rapidly enough to prevent fleas from biting and feeding as fleas feed within minutes of arriving on a host.

Other parasitic diseases

Other parasitic diseases that should be considered when investigating a pruritic cat include pediculosis, notoedric mange, cheyletiellosis, harvest mites, otodectic mange and demodicosis.

Pediculosis

Uncommon feline dermatosis caused by the biting louse *Felicola subsrostratus*. Lice infestations are more common in more northerly latitudes. The life cycle is entirely on the host and eggs are cemented firmly to hair shafts.

Clinical signs include pruritus, miliary dermatitis, eosinophilic skin diseases and in severe cases, anaemia. Diagnosis is on identification of lice or eggs on hair plucks, coat brushings or acetate tapes. Treatment is usually straightforward. Any topical spot on product that kills fleas will be effective against lice. It is not necessary to treat the environment.

Notoedric and sarcoptic mange

Contagious, pruritic disease caused by *Notoedres cati* a sarcoptiform mite. The mite has medium length unjointed stalks with suckers on the two anterior pairs of legs and a dorsal anus (terminal anus in *S. scabiei*).

Sarcoptic mange resulting from *S. scabiei var canis* is a rare disease in cats[5] but has been reported. The three week lifecycle of the mite is spent entirely on the host. Mites can survive off the host for 7 days. Clinical signs are of intense pruritus over the face, pinnae, and later the feet and perineum. Severe excoriations and self-trauma result. Eventually the skin becomes chronically thickened and crusted.

The diagnosis is based on history, clinical signs, and identification of the mites on skin scrapings and response to treatment. Unlike canine scabies the mite is easy to find on skin scraping.

Selamectin or moxidectin spot on at 4 week intervals should be effective. Alternatives include weekly lime sulphur dips[6] or ivermectin given orally or by s/c injection at 300ug/kg weekly. Environmental treatment is also recommended.[5]

Cheyletiellosis

Caused by non host-specific *Cheyletiella* mites. *Cheyletiella blakiei* in the cat, *C. yasguri* in the dog and *C. parasitovorax* in the rabbit. These are comparatively large mites that are highly contagious. The 35 day lifecycle takes place on the skin. Mites do not burrow into the skin but live in superficial pseudotunnels. The mites can survive up to two weeks off the host.

Clinical signs are variable and include pruritus, scaling, miliary dermatitis +/- alopecia.
Diagnosis is on history, clinical signs, identification of mites on coat brushings or skin scrapes and on response to treatment. In the author's experience, the mites can be very difficult to find and trial therapy is indicated if the disease is suspected but mites cannot be found.

Treatment - all in contact animals should be treated (including dogs and rabbits). Options include fortnightly or monthly selamectin or moxidectin, weekly lime sulphur dips, fipronil sprays (every 2-3 weeks?), ivermectin given orally or s/c injection 200-300ug/kg every 7 (oral)-14(injection) days.[5] All washable bedding should be washed at >55C and all other bedding discarded. The environment should be then treated with a permethrin spray.

Trombiculidiasis (harvest mite infestation)

Seasonal pruritic disease caused by *Neotrombicula autumnalis.* The adult mite lives in decaying vegetation and it is the six legged larval form that is parasitic to animals. In the UK there is a distinct geographical distribution with the disease seen predominantly on the east side of the country where there are chalky soils.

Clinical signs are of a pruritic papulocrustous eruption with self trauma, excoriations and crusting. The disease is seen from late summer until the first frost when the mites are killed.

Diagnosis is usually straightforward and confirmed on identification of the bright red spots (paprika) of the mites that may sometimes be found in Henry's pocket on the pinna or interdigitally. Pruritus may continue after the mites have dropped off making the diagnosis more challenging.

Fipronil sprays over the entire body monthly and the face, ventrum and feet weekly has been shown to be an effective treatment. Glucocorticoids may be required to control pruritus.

Otodectic mange

Caused by highly contagious, non-burrowing, psoroptiform mite *Otodectes cynotis.* Life cycle of 21 days spent on host. Can survive in the environment for short periods.

Clinical signs- predominantly a ceruminous otitis externa with “coffee grounds” discharge. The mites can come out of the ears and wander around on the skin resulting in a generalised pruritic skin disease with miliary dermatitis, ventral or flank alopecia.

Diagnosis is based on identification of mites in cerumen or on coat brushing examination and response to treatment.

Usually responsive to topical antiparasitic therapy. Most proprietary otitis externa treatments have an antiparasitic effect. Topical therapy should be applied for at least 20 days to ensure that all ova have hatched and newly emerged larval stages have been exposed to the drug. The use of a cerumenolytic facilitates exposure of the parasites to the topical therapy.

Selamectin and moxidectin given once, or repeated after 30 days, are licensed for the treatment of otodectic acariasis. Oral or injectable ivermectin 200-300ug/kg orally or by s/c injection at 10 daily intervals on three occasions would also be effective. All in contact dogs and cats should be treated.

Demodicosis

Rare disease in the UK. The long, slender hair follicle mite is *Demodex cati. Demodex gatoi,* a short tailed mite,[7] inhabits the surface layers of the epidermis.

Little is known about the immunology of feline demodicosis although underlying immunosuppressive diseases such as diabetes mellitus, feline immunodeficiency virus, feline leukaemia virus, systemic lupus erythematosus, and hyperadrenocorticism, and Bowen’s (carcinoma *in situ*) disease may be predisposing and exacerbating factors.

The disease may be localised or generalised.

D. cati infestation most commonly results in variable pruritus, alopecia, erythema, scaling and crusting over the eyelids, periocular skin, head, neck and chin and ceruminous otitis externa.[8] Regional lymphadenopathy is seen in generalised disease.

Clinical signs associated with *D. gatoi* infection include pruritus, erythema, scaling, excoriation, crusting, broken hairs and symmetrical alopecia. *D. gatoi* infestation is known to be contagious.

Diagnosed on skin scraping/histopathology.

Weekly lime sulphur dips are probably the treatment of choice for *D. gatoi* infestation.
Treatment using amitraz at concentrations between 0.0125 and 0.025% w/v although side effects of sedation, ptyalism and hiding behaviour may be seen. Weekly subcutaneous injections of doramectin at a dosage of 0.6mg/kg were reported to be curative in three cats with generalized demodicosis. Crotamiton, selenium sulphide and milbemycin oxime[9] have all been reported to be useful. The author has successfully treated two cases of *D. gatoi* infestation with daily oral ivermectin at a dosage of 400ug/kg.

Infections

Both Malassezia yeast and bacterial infections are common complicating factors in feline pruritus. Their identification and treatment is an essential part of the investigation and management of pruritus in cats. If there is clinical or cytological evidence of microbial infection, then this should be thoroughly treated.

On occasion, management of the secondary infection is key to resolution of disease. As a general rule thorough flea / ectoparasite treatment would be administered in conjunction with antimicrobial therapy. If all lesions and pruritus resolve with antimicrobial and flea therapy, then strict flea control should be maintained and the cat should be monitored for recurrence. If symptoms of pruritus remain following treatment of microbial involvement, then a dietary trial is indicated to rule out an adverse food reaction.

Malassezia dermatitis

The genus *Malassezia* comprises a group of lipophilic yeasts that are most commonly isolated from the skin and mucosae of mammals and birds. At least ten species are recognised. Cats are often colonised by *M. pachydermatis* and occasionally other species.

Cats can develop localised or generalised *Malassezia* dermatitis. Clinical signs of Malassezia dermatitis in cats include multifocal alopecia, erythema, crusting, greasy brown scales and brown greasy discharge over the nails.[10,11] Both *M. pachydermatis* and the lipid dependent species of Malassezia can be involved.[12] Localised infection can be a complicating factor in allergic dermatoses[10] and some breeds (Devon Rex[12]) are predisposed to colnisation with *Malassezia* spp. Generalised *Malassezia* dermatitis carries a poor prognosis as most cats will have an underlying immunosuppressive disease.[13] The diagnosis of *Malassezia* dermatitis is on cytological examination. The unipolar budding yeasts, stain a purple colour on cytology. In some situations only the capsule of the yeast stains and these known as ghost forms. *Malassezia* infection should be treated as part of the diagnostic work up of a pruritic cat.

- The treatment of choice is three times weekly miconazole / chlorhexidine shampoos (Malaseb®). When cats will tolerate shampoo therapy, this treatment is usually effective.

- Where shampoo treatment will not be tolerated systemic therapy would be indicated using itraconazole (Itrafungol®; Janssen) given at a dosage of 5mg/kg sid for two to three weeks.[14] Pulse therapy is also likely to be effective.

Pyoderma

Bacterial infections are a common complication of many feline allergic and keratinisation disorders but may also be a feature of underlying immunosuppressive disease. Bacterial skin disease in cats can contribute significantly to pruritus. The majority of cases of pyoderma involve *S. pseudintermedius* although the potential list of bacteria causing cutaneous infections in cats is extensive.

Clinical presentations of feline pyoderma

The clinical presentations of pyoderma in the cat are much less well defined than they are in the dog. They tend to be clinically indistinct and usually reflect the underlying disease process[15]. However, superficial or deep pyoderma may be distinguished in some cases. Superficial lesions include crusted and eroded papules, exudative plaques, erosions and ulcers, areas of non-specific self trauma, lesions of feline acne. Deeper infections include non-healing wounds, nodules and draining tracts. A common scenario is secondary bacterial infection of an eosinophilic dermatosis[16].

Treatment

It has become apparent that systemic antibiotic treatment is required much less frequently than previously thought for the management of pyoderma. Some national guidelines recommend that topical antiseptics should be attempted before resorting to systemic antibiotics provided the animal's welfare will not be compromised. In one study of feline superficial pyoderma[17], 31% of cases were treated with topical therapy alone and half of those treated had a good response. Topical therapies used included chlorhexidine, silver sulphadiazine and fusidic acid. However, there are difficulties in treating cats topically and systemic antibacterials are probably still the mainstay of treatment for feline pyoderma. Empirical antibiotic selection is acceptable in first presentation, uncomplicated infections where cocci are identified on cytology. Culture and sensitivity testing should be performed:

- If unusual organisms such as rods are seen on cytology

- If there has been a poor response to empirical therapy

- Cases that have been previously exposed to many classes of antibiotics
- In cases of deep pyoderma when more prolonged therapy may be required
- Where there is a history of exposure to a potentially resistant bacteria
- Immunosuppressed cases

Samples for culture and sensitivity testing should be taken from sites where bacteria are identified on cytology. The optimal lesion to sample is an intact pustule but any other lesion type may be sampled. Deep draining lesions should be cleaned first with dilute chlorhexidine or alcohol then material expressed from the lesion. Fine needle aspiration and biopsy procedure are effective methods of obtaining samples from deeper lesions.

Guidelines have been published on the use of systemic antibiotics for the treatment of superficial pyoderma in dogs[18] but no such guidelines are currently available for cats. Nevertheless, it is reasonable to extrapolate from the canine guidelines. These guidelines classify antibiotics into three tiers. The first includes cefadroxil, cephalexin, clavulanate-amoxicillin, clindamycin, and lincomycin. Cefpodoxime and cefovecin may be included as first line treatments where medication is difficult or owner compliance is likely to be poor. First tier products are suitable for initial empirical treatment of pyoderma. Second tier include cefovecin, cefpodoxime, difloxacin, enrofloxacin, marbofloxacin, orbifloxacin and pradofloxacin. They should not be used for empirical therapy and should only be used following culture and sensitivity testing.

Dosage and duration of therapy

The drug should be administered at the correct dose to achieve adequate tissue concentrations. Due to relatively poor cutaneous vascular perfusion, it is wise to treat at the higher end of the recommended dosage range. Most dermatologists continue treatment until a week past clinical and cytological resolution and in the case of feline superficial pyoderma this is likely to be two to four weeks. Considerably longer courses of treatment may be required for deeper infections. It is important for the clinician to re-evaluate the animal to determine the end point of treatment and not to rely on the owner's judgment.

Wherever possible the underlying disease predisposing to the pyoderma should be identified and treated.

Feline allergic dermatitis

Introduction

The variable clinical phenotype and conflicting evidence for the importance of IgE in has led to a great deal of controversy over the terminology and diagnosis of feline allergic skin disease. However, in practice the investigation of a pruritic cat is similar to the process in the dog and having ruled out ectoparasitic involvement and treated yeast and bacterial infections, the next step is to investigate the possibility of an adverse food reaction.

Adverse reactions to foods

Cutaneous adverse food reactions are an uncommon cause of pruritus in the cat that may account for between one and 12% of pruritic feline skin disease.

Not all adverse reactions to foods have an immunological basis, hence the term cutaneous adverse food reaction rather than food allergy. Adverse food reactions may be defined as:

- Adverse food reaction: aberrant reaction after ingestion of a food
- Toxic reaction: dose dependent reaction to a toxin
- Food hypersensitivity: abnormal immunologic response
- Food intolerance: a non-immunologic response

It has been speculated that type I, III and IV hypersensitivity responses maybe involved in food hypersensitivity[19] in cats and dogs but the reality is that despite the amount written about cutaneous adverse food reactions in recent years, the exact pathogenesis of this disease has yet to be elucidated. Extrapolating from human literature, most food allergens are thought to be large glycoproteins of 10-70KDa.

Clinical signs of dietary hypersensitivity in the cat

There is no age or gender predilection but the Abyssinian may be predisposed[20]. Cats may present with any of the cutaneous reactions patterns. A recent large study that showed involvement of the face was more common in food hypersensitivity dermatitis than non-flea, non-food induced allergic dermatitis.[20] Concurrent GI symptoms have been reported in between 10 and 30% of cats with food allergy. The wide variety of clinical signs means that a diagnosis of food allergy cannot be made on clinical signs alone.

Diagnosis

The only reliable method for diagnosis of a food allergy is observation of the response to feeding a selected, restricted antigen diet. This presents significant problems in cats. In the author's practice we aim to feed the diet for around four to six weeks but some cases are reported to take up to 10 weeks to respond.[21] Clinical signs should resolve when the restricted diet is fed; there should be a relapse on challenge with the original diet and then improvement again with re-institution of the restricted diet.

Diet selection

Two types of restriction diet are currently available –novel protein or hydrolysed.

Novel protein

In order to develop a food hypersensitivity, the animal must have previously been exposed to a food. Proteins are considered the most common food allergens. Thus, a novel protein diet is one containing a single protein or carbohydrate to which the animal has ideally never, or only very rarely, been previously exposed.

A novel protein diet may be fed as either a home cooked diet or a commercially prepared dried or tinned food. Although it may possibly be the case that commercial diets are inferior to home cooked diets in the diagnosis of cutaneous adverse food reactions,[22] most dermatologists now consider that improved owner compliance and elimination of concerns about inducing dietary deficiency when feeding a commercial diet greatly outweighs any potential disadvantage of possible "hidden" allergens. Additionally, many cats seem to find commercial diets more palatable.

Hydrolysed diets

The other alternative is to feed a hydrolysed diet. In theory, it should not matter what the animal has previously eaten and the benefit of hydrolysates has been demonstrated for the diagnosis of food allergies in dogs[23] and the treatment of inflammatory bowel disease in cats.[24] However, these diets will only rule out the involvement of type I hypersensitivity reactions and there has been doubt about how effective the hydrolysis process really is. The majority of peptides in a hydrolysed diet will be less than 3KDa but there will still be a small percentage of molecules 10KDa or greater[25] and partly for this reason, it has been suggested that a novel parent protein source should be used when selecting a hydrolysed diet.[26]

When undertaking a diet trial consideration should be given to the following:

1. **Concurrent infection/ectoparasitic therapy.** Thorough and rigorous flea control should be maintained throughout the course of the diet trial.

2. **Control of pruritus.** The use of short courses of glucocorticoids during the diet trial should be considered for humane reasons and to improve compliance.

3. **Compliance.** Lack of client compliance is the biggest reason for the failure of diet trials. Improved client education significantly decreases the failure rate in diet trials.[27] A careful explanation of why a diet trial is indicated, what foods may and may not be fed and identification and discussion of any concerns the owner might have are essential prior to starting a trial. Written instructions should be given and cases should be followed up after a period of two to four weeks.

Challenging the restriction diet

In most allergic skin diseases, the pruritus waxes and wanes. An improvement may due to concurrent antimicrobial treatment, a seasonal effect or merely a spontaneous resolution of pruritus. For this reason, a diet challenge with the original food is indicated following any apparent improvement. If an adverse food reaction is involved, this should result in recurrence of symptoms within seven to 10 days. If there is no increase in pruritus following the dietary challenge then a CAFR can be ruled out and the apparent improvement was due to some other effect.

Long term management

The best way of managing food allergy is to avoid feeding the causative foodstuffs. Long term, feeding commercially available limited ingredient or hydrolysed diets are the most appropriate option. Long term glucocorticoid therapy may be required if the cat will not eat an appropriate diet.

Does feline atopic dermatitis exist?

Canine atopic dermatitis has been well-characterised and is described as a "genetically predisposed, inflammatory and pruritic skin disease with characteristic clinical features associated most commonly with the development of IgE antibodies to environmental allergens".[28] This definition encompasses most of the parameters within which the diagnosis is made.

Antihistamines	1-2 weeks
Topical glucocorticoids	2-4 weeks
Depot glucocorticoids	6-8 weeks
Ciclosporin, EFA's	Don't affect tests

There is much controversy as to whether a similar disease exists in the cat. There is little evidence of a genetic predisposition and the clinical phenotype in cats with presumed atopic dermatitis is diverse. Thus, there is no characteristic clinical phenotype equivalent to that of canine atopic dermatitis. The existence of feline IgE has been unequivocally demonstrated[29,30] but its role in feline atopic dermatitis is unclear.

Feline hypersensitivity dermatitis

In view of the uncertain involvement of IgE in feline atopic dermatitis and the variable clinical picture, it has recently been proposed that the term **non-flea, non-food**, **feline hypersensitivity dermatitis** is used.[20] The diagnosis of NFNFHD is made on ruling out of flea allergy and other ectoparasitic disease, identification and treatment of infections and diet trials. It is worth noting that in this large study, 24% of 501 pruritic cats had diseases other than HD underlining the importance of a thorough work up in these cases.[20]

Intradermal vs serological testing

Despite the concerns about the involvement of IgE, most dermatologists adopt a pragmatic approach and perform intradermal or allergen specific serology testing to identify putative allergens for avoidance purposes and allergen specific immunotherapy. Intradermal testing is a difficult technique in cats. Allergen specific IgE serology is popular amongst dermatologists and many routinely do both tests. To the author's knowledge, there are no studies that have compared the response to immunotherapy based on intradermal testing versus serology in the cat although this has been examined in the dog and results are conflicting.[31]

Table 1 Drug withdrawal periods prior to IDT

Allergens

The most commonly implicated allergens in the cat are house dust mites[32] with *Dermatophagoides farinae* being the most common reaction in the UK despite the fact that it doesn't actually occur in this country. Positive intradermal and serological reactions are also seen to storage mites. The clinical relevance of this is still not clear and these may represent a cross reaction with house dust mites. Dried stored foodstuffs have been considered to be a source of storage mites allergen but a recent study showed that mite and mite protein levels in stored food were trivial in comparison to levels in the house environment.[33]

Managing feline hypersensitivity dermatitis

Managing feline hypersensitivity dermatitis can be one of the great challenges in veterinary practice. It is important that the owner understands that this is an incurable disease but that most cases are manageable.

Allergen avoidance

In theory avoidance of causative allergens ought to be a helpful strategy in managing cats with NFNFHD that have positive allergy tests. Measures that can be taken are shown in the box.
There is limited evidence of the benefit of environmental measures in controlling allergic disease in dogs, cats and man. One open uncontrolled study in dogs with house dust mite allergies did show slight benefit from efforts to reduce dust mite exposure. The method used was to treat the house environment repeatedly with benzoyl benzoate until no mite faecal matter (guanine levels) could be detected.[34] Pyriproxyfen has been advocated as an environmental method of dust mite control.[35] Pollen avoidance is nearly impossible to achieve.

Advice to people allergic to pollens includes keeping windows closed and using air conditioning,

Allergen avoidance measures

House dust mites

Use impermeable covers on soft furnishings and bedding
Keep pet's bedding areas clutter free and remove soft toys to prevent dust build up
Keep pet away from dusty areas
Vacuum the house weekly with a HEPA filter. Keep the pet outdoors during vacuuming and for one hour afterward.
Substitute laminate flooring for carpets wherever possible.
Wash bedding weekly in a hot wash (> 130 F [54.4 C]).
Reduce relative humidity in the home to 30% to 45% where practicable (dehumidifier, air conditioning)

Molds

Avoid freshly cut grass, compost heaps, grass clippings, leaf piles etc.
Reduce relative humidity in the home to 30% to 45%.
Avoid damp rooms such as basements, bathrooms, utility areas.
Keep bedding clean and dry.
Store food in a dry environment.
Wipe damp areas with a fungicide or dilute sodium hypochlorite solution (1 part bleach to 9 parts water).

minimising outdoor activities between 05.00 and 10.00, staying indoors on high-pollen days and windy days and avoiding freshly cut grass.

Allergen specific immunotherapy

Allergen specific immunotherapy (ASIT) involves the administration of gradually increasing amounts of allergens to an individual to alleviate clinical signs associated with environmental exposure to those allergens. Allergen specific immunotherapy is often used in the management of canine and feline atopic

dermatitis and is considered the treatment of choice for the management of these diseases.[36] There are no randomised controlled studies on allergen specific immunotherapy in the cat but open studies[37] have reported benefit. One study reported success rates of 60-78% in cats treated with allergen specific immunotherapy and it was considered safe and effective.[38] ASIT requires compliant owners and co-operative patients. Protocols for immunotherapy differ according to whether aqueous or alum precipitated allergens are used. Some cats with severe pruritus may require symptomatic therapy including glucocorticoids and/or ciclosporin both during induction and also maintenance. Immunotherapy should be continued for up to a year before making any final assessment of efficacy.

Symptomatic therapy for pruritus

The object of symptomatic therapy is to find a way of managing the pruritus that controls the pruritus effectively enough to give the cat a good quality of life, that does not induce unacceptable adverse effects that the owner feels comfortable with and that is affordable. Some level of compromise is likely to be required.

Essential fatty acid supplements

There is only limited evidence demonstrated efficacy of EFA supplementation for feline hypersensitivity dermatitis. Proposed beneficial mechanisms, at least in dogs, are to direct fatty acid metabolism towards less inflammatory eicosanoids, inhibit cellular activation and excretion of various cytokines and to improve epidermal barrier function.[39] The best ratio of ω3/ω6 fatty acids is unknown in cats. It has been suggested that the best way to supplement EFA's is in the diet.[40] EFA's should be administered for at least six weeks before making any decision on efficacy. They may have a synergistic effect with antihistamines and a glucocorticoid sparing effect has been demonstrated in dogs.[39]

Antihistamines

Antihistamines have been reported to be of benefit in controlling pruritus in cats[41,42] but there are no controlled trials and it has been difficult to replicate reported results in the author's practice. They are most likely to be of benefit if the skin is only minimally inflamed and there are no secondary yeast or bacterial infections. They are probably best given continuously, and before histamine release. Chlorpheniramine (2-4mg/cat q12h), cyproheptadine (2mg/cat q12h PO, hydroxyzine (1-2mg/kg q12h PO) and oxatomide (10-30mg/cat q12h PO may be tried.

Side effects to antihistamines include lethargy, GI symptoms, polyphagia, polydipsia, increased vocalisation and hyperexcitability, Use with caution in liver disease, glaucoma, urinary retention, seizures and pregnancy.

Glucocorticoids

Glucocorticoids are probably still the most consistently effective drugs in the management of pruritus in cats[43] and they are certainly the best option for short term symptomatic relief of pruritus. Cats have fewer glucocorticoid receptors,[44] are more resistant to side effects of glucocorticoids and generally require higher dosages of glucocorticoids than dogs. Apparent treatment failures are commonly the result of inadequate dosing, particularly with lesions of the eosinophilic granuloma complex. In general, rapidly active or parenteral formulations are preferred over depot GC's because there is less suppression of the HPA axis, enhanced ability to monitor and adjust the dose and less pronounced side effects.[45] Depot GC's should be reserved for cats in whom oral dosing is not possible but this treatment is associated with increased incidence of side effects (see below) and in the author's experience may not be as effective as orally administered glucocorticoid therapy for more severe disease. However, for atopic cats with a very restricted seasonal pollen allergy, the use of methyl prednisolone acetate can be highly effective and convenient for the client.

There appears to be no circadian rhythm of endogenous corticosteroid secretion in cats so time of day is not a consideration with GC therapy in cats.[45]

The author's preference when managing pruritus in cats is to start with oral prednisolone or methylprednisolone. An induction dosage is administered to bring the pruritus into remission and then tapered to a long term maintenance level.

The use of hydrocortisone aceponate spray[46] may be an alternative to the use of systemic glucocorticoid therapy for the management of feline allergic dermatitis.

Dosage and administration

Prednisolone

Induction
Cats 1-2mg/kg PO q24h (lesions of the eosinophilic granuloma complex may require higher dosages to achieve remission).
Maintenance
Cats 1mg/kg eod

Methylprednisolone

Induction
Cats, 0.8-1.6mg/kg PO q24
Maintenance
Cats, 0.8mg/kg eod
The main advantage of methylprednisolone is fewer mineralocorticoid effects than prednisolone.

Dexamethasone / betamethasone

Induction
Cats, 0.1-0.2mg/kg PO q24h
Maintenance
Cats, 0.05-0.1mg/kg every 2-3 days.

Antihistamines and essential fatty acid supplementation[39] may have a glucocorticoid sparing effect in dogs and the same may be true in cats when used concurrently.

Adverse effects of glucocorticoids

A wide variety of adverse effects are associated with glucocorticoid therapy.
Short-term side effects of glucocorticoids in cats include polyuria, polydipsia and polyphagia, congestive cardiac failure and mood alteration.

Longer-term effects include weight gain, redistribution of body fat, pot-bellied appearance, diabetes mellitus, urinary tract infections, skin and coat changes, and liver disease.

Glucocorticoids interfere with a variety of pathways that result in insulin resistance and potentially induce overt diabetes mellitus. There is a strong association between high endogenous GC levels in cats and DM (80% of cats with HAC are diabetic) and the same may be true of exogenous sources of GCs with prednisolone being shown to decrease glucose tolerance in cats.[47]

Glucocorticoids have marked atrophic effects on the skin with suppression of fibroblast and keratinocyte proliferation. Collagen production is down-regulated. Thin skin, bruising, hyperpigmentation, skin tears, medially curling pinnae and alopecia are all reported in cats with hyperadrenocorticism. Skin fragility is thankfully a rare but serious side effect of glucocorticoid therapy in cats.

Congestive cardiac failure associated with hypertrophic cardiomyopathy has been reported in a series of cats following treatment with methylprednisolone acetate.[48] Steroid hepatopathy can occur in cats; although they do not develop marked steroid induced elevations in ALP, mild to marked elevation of hepatic enzymes are occasionally seen in cats treated with glucocorticoids.[45]

Ciclopsorin

Ciclosporin (CSA) is a calcineurin inhibitor with potent immunomodulatory activity. It suppresses the production of IL-2 by T-lymphocytes, a cytokine that is important for the activation and clonal expansion of T-cells. CSA also inhibits mast cell functions, eosinophil survival, epidermal Langerhans cell, keratinocyte cytokine secretion, and late phase responses.

It has been shown to be an effective treatment for all manifestations of feline allergic dermatitis[49] including miliary dermatitis, symmetrical alopecia, head and neck pruritus and the lesions of the eosinophilic granuloma complex.

Dosage

The dosage is 7mg/kg sid and it can be given with food or on an empty stomach. Response to treatment may be observed after four to six weeks.

Adverse effects

The most frequent adverse effects were GI symptoms with vomiting and diarrhoea that usually do not require medical intervention. Other signs include lethargy, anorexia, hypersalivation, weight loss and lymphopenia. As decreased appetite and weight loss may occur, it is recommended to monitor body weight. Hepatic lipidosis is a risk in overweight cats that have significant bodyweight reduction, Treatment should be discontinued if weight loss occurs. There was no evidence renal or hepatotoxicity. There have been concerns about the exacerbation of latent toxoplasmosis infection in cats treated with CSA.[50] Experimental work has shown that cats infected with *T. gondii* prior to CSA administration failed to develop clinical toxoplasmosis after CSA administration.[51] However, cats treated with high dosages of CSA when first exposed to *T. gondii* developed toxoplasmosis. It is important that toxoplasma negative serology cats treated with ciclosporin avoid exposure (ie restrict hunting and feed cooked diets). Further work is needed to determine the frequency of exacerbation of subclinical infection in seropositive cats. Cats seropositive to FeLV and infected with FIV should not be treated with cyclosporine. Precautions relating to diabetes mellitus, vaccination, drug interactions and risk of neoplasia are similar to those in dogs.

The main disadvantage is its cost; even for a cat this could run into several hundred pounds a year.

Megoestrol acetate

The use of **megoestrol acetate** has been associated with highly undesirable side effects in the cat, including mammary hyperplasia (with possible neoplastic transformation); iatrogenic hyperadrenocorticism; diabetes mellitus with accompanying complications (hypertension, retinal detachment); acromegaly and fragile skin syndrome. However, it was and still can be a very effective treatment for managing extremely refractive pruritus in cats but it should only be used as a last resort.

Soft claws

In our practice, we have found the use of soft claws, plastic claw protectors that are glued over the claws of the hind feet to be very useful in the prevention of self trauma in severely pruritic in cats. In general they are applied just to the hind digits.

Behaviour modification

In man a link between emotion and skin disease is well-recognised.[52] In the past, many cats with symptoms of overgrooming, particularly those presenting with symmetrical alopecia were labelled with a diagnosis of psychogenic dermatitis. This was a bit of a convenient “dustbin diagnosis” because many of these cats had allergic disease[53] and the incidence of a psychogenic dermatosis as a sole cause of pruritus in the cat is considered by referral dermatologists to be extremely low. However, some individual cats are inherently nervous, hyperaesthetic, fearful or shy and such psychogenic factors may exacerbate existing pruritic skin diseases. Oriental breeds of cats may be an increased risk of developing psychogenic dermatosis.

Consideration should be given to management of stressful trigger factors when treating pruritus in cats. Stressful trigger factors include:

- New baby in household
- Moving house
- Boarding
- Hospitalisation
- Loss of companion
- New cat in territory
- New cat in household

Management

If possible the triggering factors should be identified and corrected. If this cannot be achieved then long term medication may be the only option. Drugs reported to be of benefit in the management of psychogenic dermatitis in cats are shown in the table. Some of these treatments also have antihistaminic in addition to behavior modifying effects. These treatments should be administered for four to six weeks to assess effects and should be withdrawn slowly.

Tricyclic antidepressants	Dosage	effects
clomipramine	0.25-1 mg/kg PO q24hrs	Inhibit presynaptic serotonin and norepinephrine reuptake. Block H1 and H2 receptors
amitriptyline	5mg/cat PO q12hrs	
SSRI		
fluoxetine	1mg/kg PO q24hrs	Selective serotonin reuptake inhibitor. No antihistaminic effects.

Table 2 Behaviour modifying treatments in the cat

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