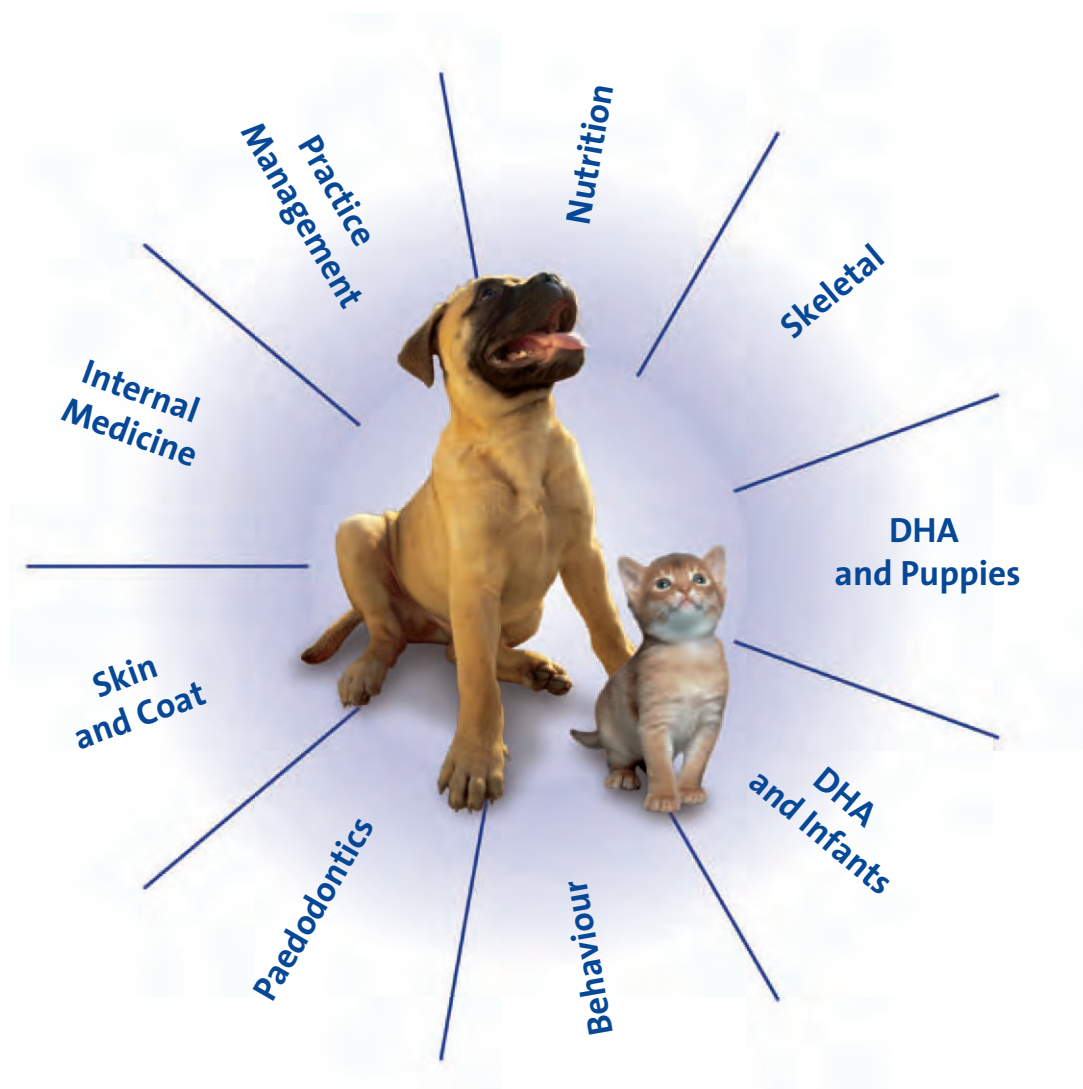


Advances in Puppy & Kitten Health Care



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SKIN AND COAT HEALTH CARE IN PUPPIES AND KITTENS

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AUTHOR'S PROFILE

Dr. Mueller graduated in Munich/Germany in 1985, completed his doctoral thesis in 1987, and worked in several large and small animal practices before completing a residency in veterinary dermatology at the University of California/Davis in 1992. He then moved to Melbourne/Australia to work with his partner and wife Dr. Sonya Bettenay in specialist practice. They established the Distance Education Programme in Veterinary Dermatology with the Postgraduate Foundation in Veterinary Science of the University of Sydney. During that time, Dr. Mueller was concurrently consulting and teaching at the Veterinary Teaching Hospital/University of Sydney. In 1999, he became Assistant Professor in Veterinary Dermatology at the College of Veterinary Medicine and Biomedical Sciences/Colorado State University. In 2004, he accepted a position as chief of the veterinary dermatology service at the University of Munich/Germany. He has published over 50 studies, articles, book chapters and books in the German, English, Spanish and French literature in Australia, Europe and America and given over 280 seminars, lectures and talks in Australia, Europe, United States, New Zealand and Canada.

THE HEALTHY PUPPY AND KITTEN

During their first months of life, puppies and kittens have special nutritional and environmental requirements to satisfy the growth and maturation they undergo during this critical period. In addition to the obvious musculoskeletal growth, their brain and immune system undergo changes influencing their future health and performance. These specific developmental requirements have been the topic of extensive research, some of which is presented in other contributions at this symposium. Some diseases which are more commonly or exclusively seen in puppies and kittens will also be discussed at this meeting. The topic of this presentation is the skin and coat of healthy puppies and kittens as well as cutaneous disorders seen exclusively or predominantly during their first few months of life, and adolescence.

SPECIFIC NUTRITIONAL REQUIREMENTS OF PUPPIES AND KITTENS

In healthy puppies and kittens (more so than an adult) a large part of the protein and energy intake is devoted to skin and hair growth. The skin is the largest 'organ' of the body. If the protein and energy intake is of insufficient quantity or quality, the hair coat will be dull and dry.

Essential fatty acids such as linoleic acid are an integral part of the stratum corneum and contribute to the barrier function of the skin. In addition, newer research in mice and humans points to a potential direct influence on the immune system particularly by omega 3 fatty acids. One such influence for example is to downregulate MHC class II expression on antigen presenting cells.

Zinc is an important cofactor of many enzymes. Especially in

fast growing dogs requirements for available zinc are increased. Chelation with plant phytates and calcium in the food complicates the formulation of a balanced diet for growing puppies. Cutaneous changes, compatible with a zinc deficiency, have been reported with generic dog food.

Thus, a balanced and complete diet is especially important in the growing dog and cat. Minimal requirements (that prevent clinical disease) for the intake of protein, fat, carbohydrates, minerals and vitamins are long established. However, requirements may vary, especially between dog breeds and individuals, particularly for vitamins and fatty acids, and optimal concentrations are not as clearly established.

GENERALISED SCALING OF PUPPIES

A mild generalised, asymptomatic scaling may be seen in growing dogs, the pathogenesis of which is unclear. These puppies are alert, active and the scaling is not associated with any other lesions or systemic abnormalities. Essential fatty acid supplementation typically produces rapid clinical improvement. Moisturizers in form of sprays or rinses also may be beneficial. This is not a life-long primary seborrhoea, and supplementation is typically discontinued when the puppy switches to the adult dietary formulation.

CONGENITAL ICHTHYOSIS OF GOLDEN RETRIEVERS

A newly recognised syndrome has been identified in the Golden Retriever in Europe and the USA. The puppies present with a generalised (tip of nose to tip of tail) seborrhoea with typically large scales. This may be clinically distinguished from the idiopathic puppy scaling by the size and degree of the scales, the breed and the poor clinical response to essential fatty acid supplementation. Definitive diagnosis requires a skin biopsy. Treatment involves supplementation with larger than normal doses of essential fatty acids, the use of moisturising shampoos, and topical applications of shampoos containing sulphur and salicylic acid, moisturisers and conditioners. Pruritus is not a clinical feature and they do not appear to be prone to secondary pyodermas.

CONGENITAL HYPOTRICHOSIS OR ALOPECIA

Alopecia (complete hair loss) or hypotrichosis (thin hair

coat) is a hallmark of specific breeds such as in dogs the Mexican hairless and the Chinese crested, or in cats, breeds such as the Cornish and Devon rex. In these breeds the alopecia is desired and is an autosomal dominant or recessive trait. However, congenital alopecia or hypotrichosis may occur as a spontaneous mutation in any breed (fig 1) and may be linked with other ectodermal defects such as abnormal dentition. Pattern baldness is a subset of congenital alopecia with delayed onset either in puppyhood or young adulthood that can be seen predominantly in Dachshunds, and Greyhounds, American Water Spaniels and Portuguese Water Dogs.

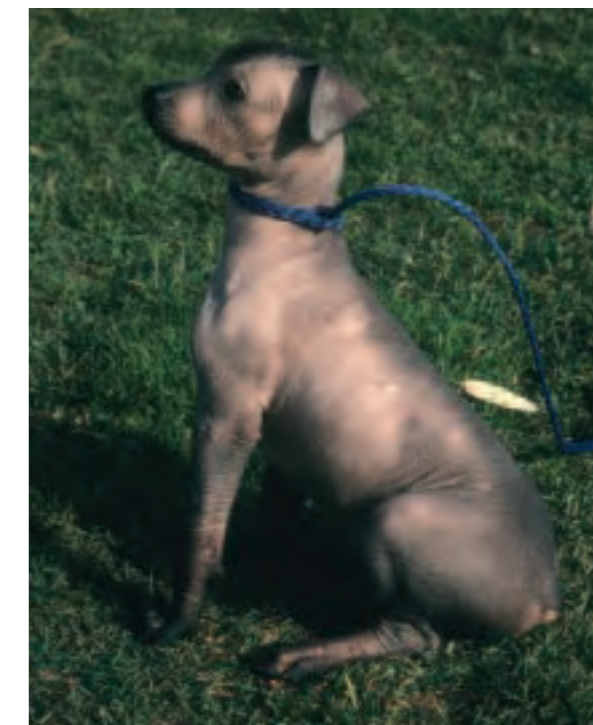


Figure 1: A young female Rottweiler puppy with congenital alopecia.

Animals with congenital alopecia are characterised by hair loss at birth or occurring within the first weeks of life. Later, the skin can become hyperpigmented and scaly. It has been reported in a number of breeds. Abnormal claw formation is also possible and attention should also be paid to the dentition, especially in dogs that have replaced their puppy teeth by adult teeth. The changes of pattern baldness are typically limited to the skin. In Dachshunds the pinnae and ventral aspects of the body may be affected, in Greyhounds the thighs are most commonly bald and in water spaniels and water

dogs hair loss occurs on the ventral neck, the caudal thighs and the tail.

Diagnosis is based on the clinical signs and histopathologic evaluation of skin specimens showing either a complete absence or severe miniaturisation of hair follicles and sometimes adnexal glands.

Treatment is limited to topical moisturisers and essential fatty acid supplementation to prevent scaling and avoidance of UV exposure to prevent sunburn.

DERMATOMYOSITIS

Familial canine dermatomyositis is a hereditary inflammatory condition of skin and muscles of young Collies, Shelties and Beauceron Shepherds. It has also been seen occasionally in other breeds. The pathogenesis is unknown. Breeding studies suggest an autosomal dominant mode of inheritance with variable expression.²

Clinical signs are evident in puppies from 2 to 6 months of age, the disease progresses variably in individual patients, but usually cutaneous lesions stabilise by 12 months of age. Lesions include alopecia, erythema, scaling and mild crusting and occur on the 'wear areas' of the face (in the periocular area and on the muzzle: fig 2), the tail and ear tips, the carpal and tarsal areas, the digit and foot pads. Claw abnormalities such as onychorrhaxis or onychomadesis may be present as well. Myositis is not always seen in mildly affected patients. With moderate disease, muscle atrophy may be observed. In severe cases, dogs have trouble drinking, chewing and swallowing food and aspiration pneumonia may occur.

Differential diagnoses include infectious diseases like bacterial pyoderma, dermatophytosis and demodicosis, congenital diseases such as epidermolysis bullosa and immune-mediated diseases such as cutaneous lupus erythematosus. Biopsies of skin and muscle and possibly Electromyography (EMG) are diagnostic tools of choice, after differential diagnoses, particularly infectious diseases, have been ruled out. Classic biopsy changes include mild interface dermatitis (apoptosis and scattered hydropic degeneration of basal cells and occasionally subepidermal clefting in the skin) and myositis with a mixed infiltrate or atrophy and necrosis of muscle fibrosis. Repeated biopsies may be required. EMG abnormalities include positive sharp waves and fibrillation potentials in muscles of the head and legs.

Trauma or extensive UV light aggravate the lesions and thus should be avoided. Prednisolone may be helpful in treating severe disease when used at high doses (2 mg/kg q24h). Pentoxifylline (10-15 mg/kg q12h) improves the perfusion of affected tissue and also decreases inflammation in other species and has been used successfully in some dogs with dermatomyositis.³ Adverse effects are rare and the drug may be combined with glucocorticoids in more severe cases. It should be given for 12 weeks before efficacy is assessed. In patients with extensive lesions, secondary pyoderma may occur.



Figure 2: Dermatomyositis in a 6 month old female Sheltie.

IMPETIGO

Impetigo is a bacterial infection (typically with *Staphylococcus intermedius*) affecting young dogs. This infection may be secondary to parasitism, viral infections, unhygienic conditions or poor nutrition, but may also occur without any identifiable cause.³

Pustules and epidermal collarettes are found predominantly in the non-haired skin of the inguinal area. Epidermal collarettes are residues of old and ruptured pustules. Neither pain nor pruritus is usually present. Diagnosis is made by clinical signs and cytologic evaluation of a pustular aspirate showing bacteria and neutrophilic granulocytes.

Lesions may regress spontaneously, particularly if an underlying cause is present and can be corrected. Most commonly local antimicrobial therapy in form of chlorhexidine shampoos or mupirocin ointment is used. Benzoyl peroxide shampoo, while effective, is frequently too irritating and drying for puppy skin. In rare case, a short course of oral antibiotics may be needed.

DEMODICOSIS

Demodicosis is an infectious, but not a contagious disease caused in most dogs by *Demodex canis*.⁴ Two other mites have been reported to cause disease, a long-bodied demodex mite⁵ that may live in the hair follicle as well as in the sebaceous duct/gland and a short and stubby one⁶ that seems to live in the epidermis. The mites are normal commensal organisms present in any dog or cat (and many other species for that matter). In some dogs, the mites proliferate causing clinical signs. Two types of demodicosis are recognised. Localised demodicosis is due to a local proliferation of mites in less than 4-6 foci (but not the paws); generalised demodicosis affects more sites or at least one paw. The reason to differentiate the generalised from the localised form in the young dog is due to the fact that the predisposition for generalised demodicosis is inherited (in young dogs, this proliferation is most likely due to an inherited T-cell defect) and these dogs should not be bred.

Clinically, canine demodicosis is characterised initially by alopecia with follicular papules. However, erythema, comedones, pustules and crusting all may occur (fig 3) and any dog with lesional skin disease should be scraped for demodicosis. Lesions can occur anywhere on the body, although the face and feet are most commonly affected. Deep skin scrapings are diagnostic in the majority of cases, although some breeds such as Shar-Peis, Scottish Terriers and Old English Sheepdogs may yield false-negative

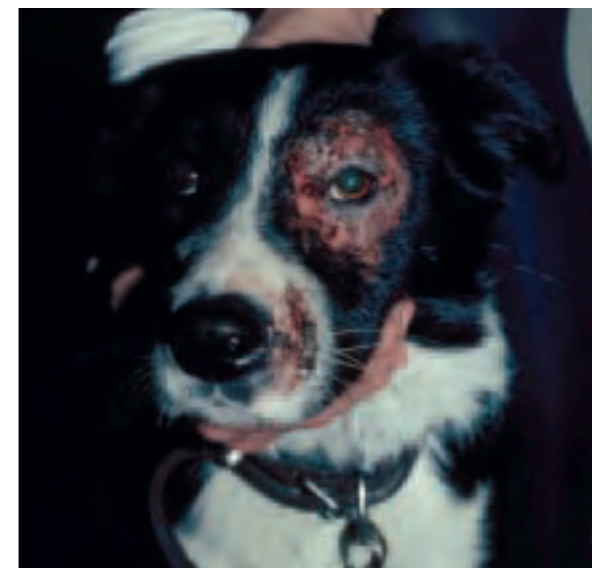


Figure 3: Demodicosis with crusting and ulceration in an 8 month old female Border Collie.

results more commonly than other breeds. Positive hair plucks may render skin scrapings unnecessary in areas that are difficult to scrape such as the eye lids, periocular area, muzzle or feet. In some rare patients, biopsies may be needed for the diagnosis.

Localised demodicosis resolves spontaneously in 95% of the patients. Treatment may be initiated against possible concurrent bacterial infections with benzoyl peroxide or oral antibiotics, but miticidal therapy is usually not necessary. Indeed, it should not be initiated in intact young dogs to identify those few patients in which the generalised form will develop and for whom neutering is indicated.

DEMODICOSIS TREATMENT

Amitraz is a formamidine and monoamine oxidase inhibitor in a xylene vehicle. The adverse reactions associated with amitraz administration resemble those induced by alpha 2-adrenergic agonists such as xylazine. These are sedation, bradycardia, hypothermia, hypotension, bloat, polyuria, vomiting and hyperglycaemia. Yohimbine at 0.1mg/kg IV antagonises the CNS-depressant and bradycardic effects of amitraz. In longer-haired dogs, clipping the entire dog is essential to allow adequate contact of amitraz with the skin surface. Crusts should be removed by shampooing with an antibacterial follicular flushing agent such as benzoyl peroxide. Whirlpooling may be beneficial. The dog has to be completely dry (2-8 hours), before being sponged down with diluted amitraz. The treating person should wear protective gloves and work in a well-ventilated area. Owners with asthma must be advised not to perform the rinses. The dog should stand in a tub with its feet in the amitraz solution to allow soaking of the often extensively affected feet. Amitraz causes a transitory sedative effect for 12 to 24 hours. Concentration of the drug and frequency of application influences the response rate. We use a concentration of 500 ppm (0.05% solution) once to twice weekly to treat *Demodex canis*. In patients with severe generalised demodicosis, the procedure should be repeated until 4 weeks after two successive skin scrapings (2-4 weeks apart) fail to reveal live demodectic mites. For pododermatitis and otitis externa, a mixture of 1 ml of amitraz with 30 ml of mineral oil can be used topically on a daily basis. Treated dogs should not get wet or be washed.

Ivermectin orally at 300 - 500 µg/kg daily is used in the treatment of demodicosis and scabies with good success. It must not be used in Collies and Old English Sheep dogs, as it commonly causes adverse reactions in these breeds. Gamma-aminobutyric acid (GABA) sends inhibitory signals from interneurons to motor neurons in nematodes and from motor neurons to muscle cells in arthropods. Ivermectin paralyzes nematodes and arthropods by potentiating GABA-binding to its receptor and stimulating GABA release. In mammals, GABA is only found in the CNS and ivermectin does not readily cross the blood brain barrier. However, in those individuals in which it does, adverse reactions include ataxia, mydriasis, tremors, stupor, salivation, bradycardia and respiratory arrest. These side effects are seen in approximately 50% of Collies at a dose between 100 µg/kg and 200 µg/kg. In our practice we have seen other breeds affected as well exhibiting ataxia and tremors at lower doses. Thus, the routine protocol for a dog, which did not previously receive ivermectin, is a slow increase over 4 days from 50 µg/kg to 100, 150 and then 300 µg/kg, which are then given daily. The owners are advised to monitor the animal carefully during that time for the above mentioned side effects. If any signs of mydriasis, ataxia or tremors occur, administration of the drug must be discontinued immediately. Note, that with daily dosing the serum level increases for many weeks due to the long half life of ivermectin, thus patients need to be monitored for side effects throughout the duration of therapy.

Milbemycin oxime is a macrolide antibiotic made from the fermentation of *Streptomyces hygroscopicus* and registered as a monthly heartworm preventative. It may be used daily at 2 mg/kg for the treatment of demodicosis. The advantages of this drug versus the conventional treatment with amitraz include the rare occurrence of side effects and the ease of administration. However, the treatment is very expensive for larger dogs. Response is comparable to amitraz. Regardless of the type of drug, treatment is continued until 4 weeks after the second negative skin scraping.

DERMATOPHYTOSIS

Dermatophytosis is a common disease of puppies and kittens. It is caused most commonly by the zoophilic fungus

Microsporium canis, but *Trichophyton mentagrophytes* (with rodents as natural hosts) and *Microsporium gypseum*, a geophilic fungus are also seen to varying degrees.⁷ In Europe *Microsporium persicolor*, a dermatophyte of voles and mice has also been reported.⁸ It typically causes facial lesions, which are not follicular. Fungal infections are usually transmitted by contact directly from animal to animal or by fomites. Contact with infected hairs and scales in the environment may also lead to clinical infection. In Europe, terrier breeds seem to be predisposed to dermatophytosis.

The classic lesion of “puppy ringworm” consists of peripherally expanding scale, crust and erythema with follicular papules and pustules (as the majority of dermatophytes grow on hair shafts). Seborrhoea, exudative nodules (dermatophytic pseudomycetoma), claw disease (onychomycosis) and depigmentation of the nostrils and planum nasale may also be seen, but those are more typically seen in adult dogs. In kittens, dermatophytosis is typically seen as focal alopecia and scaling, most commonly on the face and legs.

Diagnosis may be made by Wood’s lamp (50% of all *M. canis* strains are associated with a yellow-green fluorescence of hair shafts). However, a negative result does not rule out dermatophytosis. Trichograms of hairs plucked from the edge of affected lesions may show hyphae and arthrospores on direct microscopic examination. The most reliable method of diagnosis is a fungal culture of hairs and scale on Sabouraud’s dextrose agar. The commonly used dermatophyte test medium is not quite as reliable, occasional false negative results may occur. This dermatophyte test medium (DTM) consists of Sabouraud’s agar, with cycloheximide (to inhibit saprophytes and systemic fungi), gentamicin and chlortetracycline (to minimise bacterial overgrowth) as well as the pH indicator phenol red. Dermatophytes produce alkaline metabolites which in turn produce a red colour change. Saprophytic fungi will also utilise protein resulting in the same colour change, but colonies should be large and mature before the colour change occurs. *The colour change can only be interpreted if the colony growth is observed daily or every second day. Even then it should be used as a 'clue' and not as a final diagnosis. Final diagnosis must be made assessing the micro/macroconidia (asexual spores).*

Hyphae and conidia may be present within the stratum corneum, the follicles and in and around hairs. The number of fungal elements present is usually inversely proportional to the severity of the inflammatory response. About 80% of the patients with dermatophytes will have a positive skin biopsy.

Therapy is aimed at minimising environmental contamination to decrease the likelihood of zoonotic spread and achieving a faster resolution of clinical signs⁹, but one needs to remember that a healthy puppy or kitten typically will resolve a fungal infection without treatment within 3 months. Thus, topical treatments are most commonly used and include shampoos, creams or rinses containing azoles. Lime sulphur rinses have also been efficacious in the treatment of dermatophytosis and are safe to use for young puppies and kittens. If lesions are focal, gentle clipping of lesions with a wide margin of 6-10 cm is recommended before topical therapy begins. Care needs to be taken not to traumatise the skin and to carefully disinfect the clipper or scissors. With generalised disease, antifungal shampoos or rinses are more appropriate and should be applied twice weekly until two subsequent fungal cultures are negative. Systemic therapy is indicated with animals not responding to therapy, patients with generalised disease and multipet households or households with immunocompromised humans. Griseofulvin is the most commonly used drug and the microsize crystal form is administered at 25-60 mg/kg twice daily with a fatty meal. This drug is teratogenic and has been reported to induce idiosyncratic bone marrow suppression in cats, particularly if the patients are FIV positive. Ketoconazole and itraconazole have also been used successfully at 10 mg/kg/day. With ketoconazole, inappetence, nausea and vomiting are common. Idiosyncratic hepatotoxicity occurs rarely. These adverse effects also occur with itraconazole, although much less commonly. The latter is lipo- and keratinophilic and may be given as pulse therapy in a one week on, one week off schedule due to its accumulation in the epidermis, follicular epithelium and hair. As with topical therapy, systemic therapy should be continued until two fungal cultures are negative. However, newer evidence indicates that antifungal therapy may render false-negative cultures and an additional culture may be considered 4-6 weeks after cessation of therapy.

JUVENILE CELLULITIS

Juvenile cellulitis (juvenile pyoderma, puppy stranglers, juvenile sterile granulomatous dermatitis and lymphadenitis) is an uncommon disease of unknown pathogenesis, but dramatic clinical features. Response to high doses of glucocorticoids suggests an immune-mediated basis. Typically, puppies between 3 and 16 weeks of age are affected. Breed predispositions exist for Golden Retrievers, Dachshunds and Gordon Setters supporting a genetic component of the disease.¹⁰

Clinically, the first change is an acute and severe swelling of the face (fig 4) and or ears with submandibular lymphadenopathy. Papules and pustules may develop, followed by fistulation, draining and crusting. The pinnae are often severely thickened and oedematous. Pain and lethargy are frequently seen in more advanced cases, anorexia, pyrexia and joint pain may also be noted.



Figure 4: Juvenile Cellulitis with facial swelling, alopecia and crusting

Differential diagnoses include bacterial pyoderma, demodicosis and drug reaction. Cytology reveals a pyogranulomatous inflammation with numerous neutrophils and macrophages, bacteria are absent.

However, in some cases, severe bacterial pyoderma may develop as a secondary infection. In these patients, there are numerous microorganisms on surface cytology. Biopsy reveals severe pyogranulomatous inflammation that typically effaces the adnexal structures¹¹ and is diagnostic only in association with negative special stains and a negative deep tissue culture. For a deep tissue culture, the biopsy site is prepared surgically, and a biopsy is obtained under aseptic conditions. The top half of the biopsy is resected and the bottom half submitted for maceration culture.

Glucocorticoids are the treatment of choice. Prednisolone is administered daily at 2 mg/kg q24h until remission is achieved. Alternatively, dexamethasone may be chosen at 0.2 mg/kg q24h. The prognosis is excellent and relapses after treatment are highly unlikely. Alopecia and scarring may be permanent sequela, if the diagnosis and treatment were delayed. Secondary infections must be recognised and treated.

OTITIS EXTERNA PARASITES

Otodectes cynotis is a common reason for otitis externa in kittens and to a lesser degree in puppies. The life cycle lasts 3 weeks.

Ears are filled with the classic ground coffee-like discharge and pruritus is variable. The mite can also live elsewhere on the body, namely the tail and dorsolumbar area. Mites may be visualised on otoscopic examination, but can be identified more reliably by placing the debris gathered from the ear on a slide with some paraffin oil and examine it under the microscope.

In single animal households, local treatment with anti-parasitic ear drops for 2-3 weeks will often suffice to treat this disease. In multipet households or with recurrent disease systemic treatment with fipronil spot-on (a couple of drops in each ear and the rest of the pipette between the shoulder blades)¹² or selamectin may be indicated. Treatment with ivermectin (300 µg/kg weekly for 3 weeks for cats and dogs: see details above under 'ivermectin' for further advice on dosage for dogs) or moxidectin (0.2 mg/kg twice 10 days apart: dogs only) has also been reported as effective.

FLEA INFESTATION IN KITTENS

Young kittens with flea infestations are often presented

to veterinarians for routine health evaluations and vaccination. Depending on the flea load, affected kittens are lethargic and undernourished or even anaemic as a consequence of severe flea infestations.

A number of flea products are not registered for the use of puppies and kittens until 6-8 weeks of age. If a young kitten at 2 weeks of age is presented with severe flea infestation, physical removal of the fleas by thorough shampooing with a mild shampoo is the safest way to eliminate the fleas from the pet, but may have to be repeated regularly to maximise effect, at least, until the environment is treated appropriately. In addition, the other cats in the household need to be treated for fleas. The environment also needs to be addressed. Blankets may be washed frequently. Other fairly safe environmental control measures are borate salts for carpeted areas or insect growth-regulator containing sprays.

DIRTY FACE SYNDROME IN PERSIAN KITTENS

A hereditary facial dermatoses of Persian and Himalayan kittens may occasionally be seen. The exact pathogenesis of the disease is unclear. Especially the head, periocular and perioral areas show inflammation and adherent black exudates. Biopsy reveals severe inflammation with epidermal hyperplasia and hyperkeratosis.

Unfortunately, no effective treatment has been identified. Topical therapy will remove the exudates, but this quickly recurs after treatment is discontinued, and a long term management plan may or may not be satisfactory depending on the severity of the disease.

EHLERS DANLOS SYNDROME (CUTANEOUS ASTHENIA, DERMATOPARAXIS)

Ehlers-Danlos Syndrome in human medicine is a group of currently 10 subtypes of connective tissue diseases characterised by excessive skin fragility and hyperextensibility. Recessive cutaneous asthenia is reported in cats and dogs and characterised by soft, pliable and extensible skin (fig 5). Minimal trauma results in skin tears that typically don't bleed much and heal with scarring. Ocular abnormalities (microcornea, lens luxation, cataracts) and joint laxity may also be seen.

Diagnosis is made by classical clinical features, but skin biopsies stained with Masson's trichrome stain and electron microscopy may be useful in the confirmation of

disease. The percentage extensibility index (Vertical height of a dorsolumbar skin fold divided by body length x 100) in dogs should be greater than 15%, in cats greater than 19% for a definitive diagnosis.

Avoidance of trauma is important in management of the disease. Vitamin C has been used successfully anecdotally in dogs and cats, but scientific proof of efficacy is still lacking.



Figure 5: A cat with dermatoparaxis

SUMMARY

There are a number of important skin and coat disorders seen in puppies and kittens that warrant our attention. By obtaining a careful history and performing standard diagnostic tests most of these problems resolve satisfactorily with the appropriate therapy. Failure of the problem to improve in the expected time should lead the clinician to pursue more advanced diagnostic procedures.

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