Facial Dermatitis in Cats and Dogs

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Background

- The face is a common are for skin lesions in dogs and cats.
- This would include the dorsal muzzle, nasal planum, lip folds, medial canthus, periocular region, preauricular region, ear base, pinnae (dorsal and ventral), and head.

These regions can be involved with the following common

- Ectoparasitic conditions
  - Sarcoptic Mange
  - Notoedres
  - Demodicosis
- Immune-Mediated/Auto-immune
  - Pemphigus
  - Discoid Lupus
  - Erythema Multiforme
  - Juvenile Cellulitis
- Cornification defects
  - Sebaceous Adenitis
  - Seborrhea
  - Zinc Responsive Dermatosis
  - Ear Margin Seborrhea
  - Feline Chin Acne
  - Idiopathic Facial Dermatitis (Dirty Face)
- Infectious
  - Bacterial
    - Leproid Granuloma
    - Psoriasiform Lichenoid Dermatosis
  - Fungal/Yeast
    - Malassezia
    - Dermatophytosis
  - Viral
    - Herpes
- Allergy/Hypersensitivity
  - Atopic Dermatitis
  - Eosinophilic Granuloma Complex
  - Insect-Bite
    - Mosquito
    - Eosinophilic Folliculitis and Furunculosis
    - Fly Strike
- Metabolic
  - Hypothyroidism
  - Cushing's
- Congenital/Hereditary
  - Cornifications Defects
  - Alopecia (Non-inflammatory Alopecia)
    - Pattern Baldness
- Cutaneous Adverse Drug Reaction
  - Parasiticides
  - Anti-microbials
  - Methimazole
  - Vaccine/Ischemic Dermatopathy
- Environmental
  - Solar

    - Frost Bite (pinnae)
- Neoplasia
    - Cutaneous T-cell Lymphoma
    - Squamous Cell Carcinoma/Actinic Keratosis
    - Nodular
        - Histiocytoma/Histiocytic Sarcoma
        - Mast Cell Tumor
        - Plasma Cell Tumor

Most of the conditions presented will also be described in notes elsewhere.
The following are a selection of conditions specific to this lecture

Canine leproid granuloma syndrome

Background

- Originally reported from Zimbabwe in the 70's, in 1998 a study from 45 cases in Australia was reported (canine leproid granuloma syndrome); Similar to feline version. 1 or more nodules on head (esp. pinnae) or extremities.
- cutaneous infx w/ novel unnamed mycobacterium (likely an environmental saprophyte introduced via percutaneous inoculation via wounds or arthropod vectors- bitting flies)

Etiology

- Mycobacterium are easily visible, but never cultured; the species is not known.

Clinical signs

- Firm or ulcerated nodular pyogranulomatous lesions in dermis and SQ tissue (up to 5 cm); unlike Feline Leprosy, there is NO facial nerve involvement and no draining LNs; no systemic signs
- Nodular and ulcerated dermal and subcutaneous lesions (<5cm) primarily on the caudal aspects of the pinnae and to a lesser extent on the muzzle, face, and forelimbs--where? 84% pinna (dorsal fold) or head, other: thoracic limbs
- Lymph nodes not enlarged, and dogs are healthy otherwise.
- History
    - Who? Boxers, Boxer-cross, short-coated breeds (i.e. Pit Bulls & Staffordshire, Dobies), also GSD; 93% of dogs short coat
    - USA, Africa, Australia, New Zealand, Brazil
    - Increased incidence in fall/winter. Suggests that transmitted by biting flies

Diagnosis

- History, PE, acid-fast bacilli in direct smears/biopsy
- Histo: pyogranulomatous dermatitis and panniculitis with multinucleated histiocytic giant cells and very few to numerous bacteria; granulomatous or pyogranulomatous infiltrates; Macrophages have acid-fast bacilli inside; no clear central spaces in the granulomas unlike other Rapidly Growing Mycobacteria (Runyoun IV*)
    - Fites Faraco Stain (acid-fast mycobacterial bacilli)
    - PCR/DNA sequencing target 16S RNA gene or heat shock protein & DNA J genes (still unsuccessful, found Corynebacterineae & Propionibacterium acnes)- unlike Feline Leporsy (M. lepraemurium)
    - Cytology or histopathology revealed pyogranulomatous inflammation with few to many acid-fast mycobacterial bacilli within macrophages. The organisms could not be cultivated in vitro. DNA sequencing of part of the 16S ribosomal RNA gene region revealed 99-100% homology among fragments from five of these dogs and fragments from dogs in the south Pacific.
        - DDX: other mucobacteria, etc. (sterile pyogranuloma syndrome, MCT, histiocytoma, lymphoma)

Treatment

- Surgical excision is curative; spontaneous resolution has been reported (weeks-months); Medical management: dpxycycline or amoxi/clav reported to be effective in Aussie study; doxycycline 5-10mg/kg PO bid +/- rifampin 10-15mg/kg PO q24hr; doxycycline 5-10mg/kg PO bid +/- clavamox; floroquinolones, clarithramycin, etc.

Psoriasiform-lichenoid dermatosis of english springer spaniels

Background

- Hyperkeratosis; may not be a true keratinization defect
- Congenital/hereditary
- A similar lichenoid tissue reaction in response to a staphylococcal infection was suspected in this case. Psoriasiform lichenoid dermatosis has been described in other breeds (see below two comments/recent studies):
- Werner (2003-JAVMA) described three dogs of different breeds treated with microemulsified cyclosporine A that developed an antibiotic-responsive psoriasiform lichenoid dermatitis (Also resolution occurred with lowering the dose of CSA). A staphylococcal infection was suspected and all three cases responded to antibiotic treatment.

- Studies in human psoriasis have shown the potential of bacterial superantigens to trigger psoriasiform dermatitis (Boehncke et al., 1997).
- In a recent case report (19 month old Shih Tzu), there was rapid resolution of the skin lesions during antibiotic therapy would be more consistent with staphylococcal infection than with spontaneous resolution of the lesions—this re port showed that other breeds can be affected by this condition documented in ESSP. (Van Pouc ke, S. Vlaams Dier ge nees kun dig Tijd schrift, 2004, 73)

Cause/Pathogenesis

- Genetic predilection suggested
- +/- distinct & exaggerated reaction to superficial staphyloccal infection (Burrows et al, 1994)

Clinical features

- Young ESSPs (4-18 months), both sexes
- Asymptomatic—generally symmetrical, erythematous, lichenoid papules & plaques initially on the PINNAE (external ear canal) & inguinal region → spread to face, ventral trunk, and perianal area
- Asymptomatic, symmetric, erythematous, lichenoid plaques and papules are noted on the concave portion of the pinna, in the external ear canal, and in the inguinal region.
- Hyperkeratotic, and spread to involve the face, ventral trunk, and perineal region

Treatment

- Steroids, vitamin A, cephalosporin (but if the cause, taper dose or stop concurrent ketoconazole)

Mosquitoes—Mosquito bite dermatitis/hypersensitivity

- Early clinical signs-pruritic; erythematous plaques/papules, erosive or ulcerated, necrotic or crusted, crusting erosive dermatitis- bridge of the nose, crusted papular lesions- pinnae (convex side), Rarely- hyperkeratosis and swelling of the pads and eyelids;
- Late clinical Signs-pigment changes (leukoderma), alopecia and scaling,
- Diagnosis- physical examination, clinical signs, history,biopsy (eosinophilic dermatitis and mucinosis, eosinophilic microabcsess, nodular eosinophilic granulomas with collagen flame follicles, eosinophilic mural folliculitis; Positive IDT- mosquito extracts; Differential diagnosis: PF, PE, dermatophyte, atopy, CAFR, herpes, eosinophilic granuloma, neoplasia, other;
- Treatment: lesions resolve if cat is kept away from mosquitoes, glucocorticoids, insect repellents

Dermatophytosis (Ringworm)

- Background: Infection of keratinized tissues, claw, hair, stratum corneum; (invade and maintain selves in "keratinized" tissues), Contact with infected hair or scale; fungal elements on animal, environment, or fomites (combs, brushes, clippers, bedding, cages), SPREAD BY: cats, fleas, mites (Cheyletiella), etc; Etiology: Microsporum canis: hair shafts with arthrospores can survive/infectious > 18 months, culture from dust, heat vents, etc , SOURCE: usually infected cats!!!; Microsporum gypsum:SOURCE: soil; Trichophyton mentagrophytes:direct or indirect from reservoir, Hosts & SOURCE: rodents and rabbitsClinical Signs: Location-face/pinna/paws/tail; Age- especially young or at risk; Signs-Hair Loss (#1), +/- Scaling, +/- Mild Itching*
- Diagnostics: Culture, Biopsy, Direct Examination of Hairs: Dermatophytosis, Potassium Hydroxide Preparation (KOH)- Dissolves keratin but not hyphae walls; Wood's Lamp: Filters UV light thru cobalt or nickel glass filter; 50% of M. canis fluorescence; Allow to warm-up 5 minutes; Expose hairs 3-5 minutes; Look for "yellow-green": Tryptophan metabolites-"apple-green glow"- if actively growing (Anagen); within hair shaft at base; False Negative: iodine; False Positive: topical medications, scales, bacteria; Skin: if skin lights: Rule-out: *M. Persicola*
- Fungal Culture: Sampling: alcohol area/hemostat; pluck hair at periphery; try and get roots or Mackenzie: brush cat for a few minutes and then in-bed in culture
- Fungal/DTM Culture: DTM "Dermatophyte Test Media" Fungassay® (synbiotics) ½ of the Sab-Duet® or ½ of the Derm-Duet® (Bacti-Labs), Modified Sabourauds destrose (DTM= SAB + inhibitory drugs), sugar, inhibits growth of bacteria and saprophytes, Starts-out a golden-yellow and turn RED when dermatophyte is present (dermatophytes use protein in the media and produce alkaline metabolites, Cytology: (lactophenol blue); Fungal/DTM Culture; Sab-Duet® = DTM + Sabourauds-SAB= better fungal sporulation and colony morphology; Derm-Duet® = DTM + Rapid Sporulation Media (RSM)- turns blue
- Treatment: may require a long course; can self-cure cats (60-100 days) & dogs (2 months); duration: 6-8 weeks, cats show resolution of lesions within 3 weeks of systemic treatment; long if onychomycosis; must tx all dogs/cats in contact & treat environment; Dogs- continue 2-4 weeks past clinical cure and negative culture; Cats- continue until clinical cure & 2-3 negative toothbrush cultures 1 week apart;
    - Common topical therapy**:** Lime Sulfur Dip: Apply 1-2 times per week for 4-6 weeks; May need to be clipped first, Miconazole, Ketoconazole, Enilconazole (Imaverol®): Dip (Available in Europe—labeled for dogs and horses)
    - Common oral therapy**:** Itraconazole (Sporanox® by Janssen Pharmaceuticals- 10mg/kg/day), Fluconazole (Diflucan®, Pfizer; generic available- 10mg/kg/day), Terbinafine (Lamasil® Novartis- 20-30mg/kg/day), Griseofulvin (Fluvicin®)
    - Environmental management**:** Enilconazole, Sodium hypochlorite (Chlorine Bleach): mix 1 oz to 1 gallon of water, Virkon-5® (Farnam), Trifectant® (Vetoquinol), Vent cleaning, Curtain cleaning, Rug cleaning, Separate positive and negative cats (cultures)

- Herpes facial dermatitis Background-FHV-1 associated commonly with upper respiratory tract infections → classical rhinotracheitis +/- conjunctivitis and keratitis +/- sinusitis +/- glossitis +/- neonatal disease +/- abortion and rarely → ulcerative and crusting dermatitis; Acute, chronic, or latent infection, NO association with FeLV, FIV, or vaccination status, Corticosteroids may make the lesions worse, Cheetahs and domestic cats; Etiology-Feline herpes virus type 1 (FHV-1); double-stranded DNA virus; replicates in nucleus of host cells → intranuclear inclusion bodies , Establish latency in the trigeminal ganglia (80% have latent infection) → centrifugal axonal spread +/- direct innoculation of the skin +/- secretions (nose, mouth, eyes—corneal sequestration), Concurrent or possibly latent viral infection + stress → predispose to facial dermatitis/stomatitis; Clinical Signs-Usually 5-8 years of age, Ulcerative dermatitis of the nasal planum or haired skin of the face (erythema, serous crusting, erosive to ulcerative); pruritus suspected (secondary to infection/self-trauma), Possible proliferative and ulcerative stomatitis; Diagnosis- history (upper respiratory tract infection when younger), clinical signs, physical examination, biopsy: usually severe eosinophil-rich necrosis (cytokines chemoattractant for eosinophils) +/- neutrophilic (rare) and epidermal ulceration with extension of necrosis into hair follicles and underlying dermis; intranuclear inclusion bodies in the surface epithelia and adnexal epithelia, Immunohistochemical stain for FHV-1 (brown stain; Avidin-biotin-peroxidase-complex), PCR (DNA) testing available, Differential Diagnosis-Eosinophilic granuloma, eosinophilic ulcer, mosquito bite hypersensitivity, dermatophytosis; Treatment: Interferon omega (IFN-ω) or interferon alpha (INF-α), multiple antiviral, antiproliferative and immunomodulatory activities -inhibit cell growth → prevent replication of some viruses , induce apoptosis in virus-infected cells and enhance expression of major histocompatibility complex (MHC) class I proteins → promote CD8+ T-cell responses → stimulate cytotoxicity of natural killer cells, induces a form of nitric oxide synthase (iNOS2) and MHC class I and II proteins → important roles in immune responses to infections; Lysine 250mg – 500mg PO BID; antagonism of the growth-promoting effects of arginine, which is an essential amino acid for herpes; Manage secondary infections
- Dirty face syndrome (Idiopathic Facial Dermatitis): Background-Persian and Himalayan Cats, Suspected to be a keratinization defect possibly associated with the concentration of sebaceous glands on the face and over-production of sebaceous material, Congenital/hereditary, Etiology-Unknown; Clinical Signs- Median age of onset is 12 months, Black waxy scale exudate affects periocular, perioral, & chin and neck; become bilateral & symmetrical involving perioral area and nasolabial folds, +/-bilateral mucous conjunctivitis & submandibular enlargement, Black exudates on the distal portions of hair shafts (inflamed sub-adjacent skin); Diagnosis- Clinical signs, physical examination, history, Differential Diagnosis, Herpes, allergies, dermatophytosis, auto-immune, other; Treatment: Manage secondary yeast and/or bacterial infection, Daily cleaning, Cyclosporine--anti-proliferative effects on skin cells (keratinocytes), which is how this works for this condition; Often partial response to antimicrobial medications, Gentle removal of the dry crusted material with warm gauze or cloth +/- topical medicated wipes. Sometimes ointments are used. Salacylic acid pads may be purchase over the counter and may be helpful. This breaks-down the debris.
- Feline chin acne: Background-"Feline acne" is a "grab bag" term used to describe what appears to be a multitude of skin diseases that affect the chin of the cat; Male:female (50:50), Etiology-Idiopathic disorder of follicular keratinization (possibly associated with poor grooming, seborrheic predisposition, abnormal sebum, hair cycle influences, stress, viral, immunosuppressed, etc.); predominante free sterols, ceramides, free FAs (epidermal lipids instead of sebaceous like humans, thus sebcaeous glands unlikley major role), Clinical Signs-"Classic" feline acne: follicle plugging in the chin & lip margin region, Dark blue comedones & crusts on chin (early) → papules & pustules (middle) → severe accumulation of follicular debris → suppurative folliculitis (late) → bacterial furunculosis/cellulitis (Pasturella, B-hemolytic Streptococcus sp., Staphylococcus sp., Malassezia, dermatophyte)- (severe); +/- cysts & scarring, Chin (sometimed edematous- Jay Leno Chin= "Fat Chin"- rule-out eosinophilic granuloma w/ collagen flame figures) or face, Diagnosis-Clinical signs, physical examination, history, rule-out the possible differential diagnoses, biopsy- follicular keratosis, plugging, dilatation- comedo; folliculitis/furunculosis, pyogranulomatous dermatitis, Differential Diagnosis- Bacterial dermatitis, allergic dermatitis and Malassezia dermatitis (usually secondary to allergies), idiopathic facial dermatitis, dermatophytosis, contact irritant, demodicosis, discoid lupus erythematosus, pemphigus foliaceus, and eosinophilic granuloma (may or may not be related to underlying hypersensitivity); Management: bathing, topical (e.g., mupirocin 2%, benzoyl peroxide, etc.), systemic antimicrobials, other (retinoids, corticosteroids)
- Eosinophilic granuloma complex: Eosinophilic Disease (ED) Consists of three controversially similar/overlaping diseases (best referred to as 'eosinophilic disease'): Indolent Ulcer ("Rodent Ulcer") (IU); Eosinophilic Plaque (EP); Eosinophilic Granuloma (EG): Background-Presence of eosinophilic degranulation products coat but not alter the collagen, Inflammatory reactions of the skin, often associated with hypersensitivity/allergic diseases, but always look for underlying cause, Recently hypothesized that the Felis domesticus allergen I (Feld I), the cat allergen contained in feline saliva and hair, may → auto-sensitize cats, Eosinophils present in other conditions as well: pemphigus, neoplasia, and pyoderma (perform a biopsy)
- Indolent ulcer: Etiology-70% hypersensitivity: refractory atopy, food hypersensitivity, flea hypersensitivity +/- infection, 30% other Causes; Clinical Signs-Ulcerative mucocutaneous lesion, well circumscribed shiny red-brown-yellow, glistening, painless, NON-pruritic lesion +/- submandibular lymphadenopathy, upper lip ("dished-out"), near midline, +/- lower lip, Differential Diagnosis-Infectious ulcers (bacterial, fungal, FeLV, FIP), trauma, neoplasia
- Eosinophilic plaque: Etiology-90% have underlying allergic (i.e. FAD) hypersensitivity (Younger cats); Clinical Signs- Moist raised erythematous papules (coalesce) → orange to yellow plaques, sharply demarcated lesions, cobblestone appearance most/glistening surface (full of eosinophils and mast cells), notice white collagen specks; intensely pruritic +/- peripheral lymphadenopathy, ventral abdomen or flank, medial thigh (sometimes), Diferential Diagnosis--Infectious ulcers (bacterial, fungal, FeLV, FIP), trauma, neoplasia

- Eosinophilic granuloma: Etiology-“Collagenolytic (not really) or linear“; Clinical SignsLinear (2-4 mm wide) raised yellow lesion; papular to nodular configuration; NON-puritic, not uncomfortable, chin, mouth (surface or tongue/oral cavity/palate), rear caudal thigh, bridge of nose, pinnae, perianal, foot pads/paws (e.g., cat litter reaction) 1 or 2 feet= ulcerative/alopecic), Subset of this disease: associated with mosquito bites (nodules +/- ulceration on the face, pinnae, and footpad), “Feline chin edema" (lower lip swell-Jay Leno), differential Diagnosis, Infectious ulcers (bacterial, fungal, FeLV, FIP), trauma (SCC, fibrosacroma in oral cavity), neoplasia, plasma cell pododermatitis (feet), xanthoma (feet); chin acne (chin),

Diagnostics for eosinophilic disease

- Cytology: determine bacteria burden (swab, aspirate, impression) vs. eosinophils
- Culture and Sensitivity: punch biopsy; Bloodwork (CBC): look at blood composition (blood and tissue eosinophilia common in granulomas and plaques)
- Virus Testing: FeLV and FIP status (FeLV and FIP +/-), Dermatopathology: normally with flame figures formation Ulcer: hyperplastic ulcerated superficial perivascular to interstitial dermatitis with fibrosing dermatitis, eosinophils, neutrophils, and mononuclear cells predominating; Plaque: spongiotic (intracellular edema of the spongy layer - malpighian- layer of skin) microvesicular epidermitis (superficial) with massive dermal (deep) eosinophilia +/- microvesicles and microabscesses in epidermis
- Granuloma: palisading granulomatous dermatitis with multifocal areas of collagen coated with the released substances from degranulated eosinophils (formerly known as 'collagen degeneration‘ or ‘collagenolysis’); eosinophils common in facial/oral biopsies
- Management of Eosinophilic Disease: Control/manage allergies (flea preventatives, food trial, immunotherapy), Systemic Antibiotics and high dose cotisone
- Systemic Glucocorticoids, Cyclosporine (Atopica®)- 25mg PO (5-10 mg/kg PO/day); rule-out toxoplasmosis (IgG or IgM titers); synergystic with azole medications, doxycycline, etc.; semiannual CBC, Chemistry, and urinalysis, Griseofulvin, Gold Salts, Chlorambucil, INF-alpha, Laser, Photodynamic Therapy