Diagnosing and Treating Canine Exfoliative Dermatitis

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Canine exfoliative dermatitis (CED) (scaling and crusting disease)

The mitotic rate in the basal layer increases → overall transit time decreases →more cells are sloughed/shed from the surface. Conditions with only hyperkeratosis will not be discussed, but many conditions have both hyperkeratosis and exfoliation.

CED is a response to many causes including:

- drug/vaccine reactions
- infectious (e.g., dermatophyte, bacterial, yeast)
- immune-mediated (e.g., pemphigus)
- systemic disease (e.g., metabolic)
- malignancy
- congenital/hereditary (e.g., icthyosis)
- idiopathic
- nutritional (e.g., zinc)
- ectoparasites (e.g., sarcoptic mange)
- allergy

Cornification

Complex & highly organized end product of epidermal differentiation that results in the formation of a structural barrier through which neither hydrophilic nor hydrophobic molecules (primarily) lipids can easily pass

Normal patterns

- Compact
- laminated
- Basket weave

Convergence of (3) separate synthetic pathways followed by desquamation (catabolic process): Formation/organization of keratin intermediate filaments keratin- an intermediate filament (smaller than microtubules and larger than microfilaments)→ all make-up cytoskeleton; keratinocytes has the most & most diverse

Keratinization

synthesis & expression of different keratins assembled in microfilaments by fillagrin (protein matrix); cornifed envelope is made-up of involucrin, keratolinin, loricrin, filaggrin; form lipid morter (from lipids and corneocyte enzymes=> cohesion)

Keratinization defects

result of anything altering proliferation, differentiation, and desquamation etc. +/- epidermal lip formation & deposition alterations

- Lipid Mortar= main barrier for water loss (ceramides + hydroxyacids= lamallae between cells + FFAs= intermediate layer)
- Lamellar Bodies= essential part of the barrier & prevent water loss
- Intercellular Lips= important part of SC barrier
- Sphingolipids
- Free Sterols
- Free Fatty Acids
- skin film is made-up of emulsion derived from epidermal degredation products and sebaceous gland secretion
- sebum= sterols (cholesterol), waxes (diesters), & FFAs (from hydrolysis of lipids by saprophytis bacteria on skin)

Xerosis (Dryness)

caused by decreased water content (Normal is when > 10% of skin is water); moisture loss occurs through evaporation to environment under low humidity & must be replenished from lower epidermal & dermal layers

Xerotic skin: Thickened SC, disorganized, fissured

- Diseased state
  - increase FFAs & Cholesterol & decreased waxes → leads to increased pH (N= 5.2-7.2) and thus increased bacteria → skin infection' also since increase in FFAs → OKH along with increased epidermal turnover

Sebaceous gland secretion influenced by

- hormone
- hereditary
- intrinsic factors (linoleic acid, vitamin A, vitamin E
- environment (temp/humidity)

(3) Classical types/forms of seborrhea/seborrheic skin conditions

- Dry Seborrhoea (sicca)
  - e.g., cheylitella
- Greasy Seborrhoea (oleosa)
  - e.g., vitamin-A responsive dermatosis

- Seborrheic Dermatitis

(2) Disorders of Hyperkeratosis (Not keratinization defects)

- **Retention Hyperkeratosis**: corneocyte desquamation caused by chemical changes to lipids/cement; i.e., sebaceous gland destruction (i.e. sebaceous adenitis), also leishmaniaseis
- **Proliferative Hyperkeratosis**: surge of epidermal activity → defective keratinocyte maturation (e.g., allergy, parasite)

Types of scaling (often associated with these defects): Scale: shedding dead epidermal cells/stratum corneum (exfoliative pattern)

- Psoriasiform- large & relatively thick scale (e.g., CTCL, EM, cyclosporine over dose, cheyletiellosis, leishmania)
- Pityriasiform- small & thin scale (e.g.,allergy, demodex, color dilution alopecia, etc.)

Key Features of Keratinization Defects: Can be generalized or focal; Secondary seborrhea is most common; therefore look for the primary disease; True keratinization defects are rare: Idiopathic seborrhea, sebaceous adenitis, ichthyosis, epidermal dysplasia, Schnauzer comedo syndrome- DDX: Folliculitis, autoimmune skin disease, zinc responsive dermatosis, Other causes of secondary seborrhea (endocrine, allergies, infections, nutrition)

Selected conditions (Some described in other lectures)

Seborrhea

- Primary Seborrhea
- Secondary Seborrhea

Background

Chronic skin reaction pattern in the skin characterized by a defect in keratinization with increased scale formation, excessive greasiness of skin/hair, & +/- inflammation (often with secondary infection—yeast/bacteria)

- Sicca- dryness of skin, increased white scaling non-adherent scales, dull/dry
- Oleosa- moisture of skin, increased keratosebaceous, malodorous debris
- Dematitis- scaling + greasiness w/ gross evidence of local or diffuse inflammation; multiple discrete to coalescent, scaly or crusty pruritic patches
    - Note: this classification system cannot be used to direct the diagnostic effort to find a cause, but help in selection of topical therapy
    - Often associated with bacterial and yeast infections (Malassezia- lipolytic nature of the organism worsens any already greasy condition; increase proliferation rate of keratinocytes (thus scaling)—thus, initial seborrheic condition encouraged yeast overgrowth & then the yeast stimulated the seborrheic change

Can be localized or generalized

- Localized seborrheic dermatitis: circular lesions with alopecia, erythema, marginal epidermal scaling, and later hyperpigmentation (e.g., ear margin seborrhea)

Primary versus secondary seborrhea

- **Primary seborrhea**
    - Inherited disorder of epidermal hyperproliferation; inherited disorder of keratinization or cornification
    - Uncommon
    - Canine Primary Seborrhea: (Cocker Spaniels)
        - Basal cell labeling indices are 3-4 x's > than normal values
        - Epidermis, hair follicle infundibulum, & sebaceous glands were hyperproliferative [the hair root matrix was normal]
        - Epidermal cell renewal time = 8 days (N= 21 days) (Cockers & Irish Setters)
        - Unknown cellular defect that leads to this (will remain hyperproliferative in cell culture or skin graft site)
        - Autosomal recessive trait in WHWT (Raczkowski, 1984); WHWT have epidermal hyperproliferation (epidermal cell renewal time studies not performed)
        - Occur early & progress as age (i.e., 10 weeks old in WHWT); usually dogs present 12-18 months of age
        - Clinical Signs: ceruminous hyperplastic OE; dull hair coat with excessive flaking/scaling of skin; greasy (sometimes) malodorous skin- esp. body folds; follicular casts; multiple discrete to coalescent, scaly or crusty pruritic patches (seborrheic dermatitis); digital hyperkeratosis & dry-brittle claws +/- pruritus+/- secondary bacterial and/or yeast infections
        - Location: around the eyes & mouth, on pinnae, skin folds, feet, axillae, groin
        - Breeds:
            - Sicca: Doberman Pinchers and Gordon/Irish Setters
            - Oleosa: WHWT, Cockers***, Springers, Labs, Bassets, Shar Pei
        - DX: by exclusion of other underlying factors/causes
            - DDX- demodex, cheyletiellosis, nutrition, ichthyosis, epidermal dysplasia, food allergy, etc
            - HISTO: hyperplastic superficial perivascular dermatitis, OKH or PKH, follicular keratosis, +/- apoptosis of keratinocytes; (mild inflammation)- plasma cells & lymphocytes, (inflammed)- ^superficial perivascular dermatitis, papillomatosis & focal areas of PKH (caps) over edematous dermal papillae (papillary squirting); +/- EH

        - TX: see topical therapy lecture notes, manage secondary infections, retinoids, cyclosporine, vitamin A, Calcitriol, etc.
- **Secondary seborrhea**
    - Caused by some external or internal insult that alters the proliferation, differentiation, or desquamation of surface & follicular epithelium
    - Common- see selected conditions discussed

Ichthyosis (Fish scale disease)

- Form of primary seborrhea-abnormal desquamation (excessive flaking)
- Greasy malodorous skin, itching, and other skin problems.
- Skin (epidermis) and hair follicle/glands (sebaceous glands) are very over-active (hyperproliferative); excessive scaling; nose and footpads may be thickened (hyperkeratotic); skin cells turn-over at a much higher rate than normal (e.g., 8 days compared to every 21 days); secondary infections (yeast and bacteria).
- This is a rare congenital skin disease in Golden Retrievers(AR) and the American Bulldog. Other: West Highland white terrier, Cavalier King Charles spaniel, Doberman pinscher, Jack Russell terrier, Norfolk terrier, and Yorkshire terrier. Biopsy confirms a diagnosis.
- Treatments.

The secondary infections are managed

Topical therapies: shampoos and sprays - moisturize (lactic acid, propylene glycol, etc.) the skin and normalize the skin and break down scaling (sulfur and salicyclic acid or tar).

Oral therapies include essential fatty acid supplementation, vitamin A therapy or retinoid therapy (isotretinoin), vitamin D analogs (calcitriol, 10ng/kg/day), corticosteroids (decrease cell turn-over/inhibit replication/side-effects long-term), chemotherapy medications, and possible cyclosporine (normalize skin cells).

Leishmaniasis

- Old World: Mediterranean basin, Portugal, France, Germany, Switzerland, Netherlands, S. Russia, India, China, E. Africa
- New World: S. America, C. America, USA (TX, OK, OH, Michigan, AL, MD)
- Affects dogs usually < 5 years
- Overrepresents: Dobermans pinchers & German Shepherds

Canine (Cutaneous and Visceral) Leishmaniasis (Leshmania sp.) is a chronic parasitic disease that is transmitted by blood sucking sand flies (Lutzomyia or Phlebotomus) → ingests blood from an infected animal (amastigotes) → inside the fly intestine, there is a transformation/maturation process (paramastigotes and promastigotes) → Leshmania migrate (have flagellum/"tail") to the mouthparts→ fly then serves as the "vector/intermediate host" and transmits the infectious agent to the host (human/dog) → parasite (protozoa) is than engulfed by certain cells in the skin (Langerhans cells) → parasites are transformed back into the amastigote form (the "classic" part of the cycle detected in the cells) 2-5 um oval bodies with single nucleus/kinetoplast)

Cutaneous signs

- non-itchy dry scaly (exfoliative) dermatitis
- alopecia

Location: pinnae, periocular, muzzle, head, and back may be affected

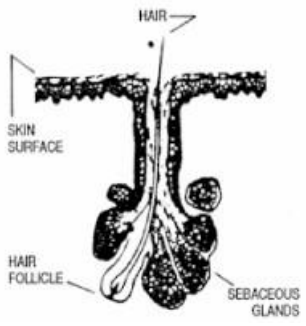
Ulceration, especially over pressure points (ischial tuberosity, elbow, hock/ankle, foot pads), pinnae, nostrils, genitals, and mouth. There can be signs of bruising (petechia/ecchymosis) of the skin and especially the penis (if applicable)

Lichenification

Classically a thickness, alopecia, and hyperpigmentation around the eyes (lunnetts). The nails may be deformed and long (onychogryposis).

There can be cutaneous nodules as well. About ¼ of affected dogs also show ocular effects (uveitis, dry eye, blepharoconjunctivitis, etc.).

- Systemic CS: lethargy, PU/PD, D+, pyrexia, weight loss, generalized lymphadenopathy, hepatosplenomegaly, muscle atrophy, lamness, epistaxis
- Immunocompromised dogs are more at risk as the body's natural defenses (e.g., phagocytes kill engulfed parasites) are weakened. There may be abnormal production of immunologic substances (antibodies), leading to more problems (vasculitis, polysrthritis, cutaneous ulceration, uveitis, glomerulonephritis).
- Systemic signs (pale gums from anemia or red-brown from liver/kidney damage (ascites), bleeding from the nose/epistaxsis associated with low platelets vs. vasculitis (immune-complexes) vs. ulceration, anorexia, depression, weight loss, increased drinking/urinating, diarrhea, vomiting, arthritis and/or myositis (lameness), liver and or kidney involvement, enlarged spleen, enlarged lymph nodes) associated with this condition.
- Lab work, including: anemia (poorly regenerative) +/- low platelets, increased white blood cells, increased globulins (polyclonal gammopathy, beta and gamma globulins) and normal or low albumin, increased BUN/Cr/protein in urine (kidney), increased ALT/AST (liver), serology
- Treatment: meglumine antimoniate (Glucantime®) and sodium stiboluconate (Pentosam®) +/- Aminosidine; and Allopurinol



Sebaceous Adenitis

Background

Sebaceous adenitis is a skin condition in which the sebaceous glands (glands associated with hair follicles/responsible for moisturizing skin) become inflamed for numerous potential and/or unknown reasons, and are eventually destroyed.

This eventually leads to the progressive loss of hair. This is believed to be hereditary/genetic or auto-immune in some cases. The sebaceous gland normally produces a fatty secretion (sebum) that helps to prevent drying of skin (lubrication) and protect from infection (anti-microbial properties).

If or when the sebaceous glands are no longer functioning properly or destroyed, the patient may exhibit a variety of symptoms. There is plugging of hair follicles and scaling that is due to the complications of reduced sebum flow due to the sebaceous gland inflammation and destruction.

Pathogenesis

Sebaceous adenitis may be a primary keratinization defect (primary cornification defect of follicular infundibula, glands, ducts), secondary keratinization defect (clogging of the ducts), inflammatory disease of sebaceous glands ("foreign" body inflammatory response), a result of decreased production of sebaceous lipids associated with sebaceous gland destruction (lipid metabolism abnormality), idiopathic (granulomatous) disease, or some auto-immune response against sebaceous glands.

Clinical signs

Clinical signs most noted include: loss of hair (alopecia), thickening of the skin (lichenification), darkening of the skin (hyperpigmentation), scaling of the skin (dandruff), secondary skin infections (bacterial and/or yeast), and often malodorous skin and/or ears.

There is visible debris adhered to the actual hairs (follicular casting). The initial signs often start on the face/head/ears with moist and exfoliative/scaling lesions. There is rarely itching associated with the disease (except for association with secondary infections).

Signalment/presentations

Sebaceous adenitis is most often seen in the Standard Poodle (hereditary- autosomal recessive), but it is also commonly diagnosed in Vizslas, Akitas, Samoyeds, Weimaraners, Old English Sheepdogs, Dalmatains, German Shepherds, Miniature Pinschers, Collies, Lhasa Apsos, and Golden Retrievers. The condition has also been diagnosed in numerous other breeds (> 55 breeds) and mixes. Dogs usually present between young adult to middle age (1-5 years of age). There are two major presentations. The first is seen in long-coated breeds (e.g., Standard Poodle, Akita, Samoyed, Belgian Sheepdogs, and German Shepherds) and the second type is seen in short-coated breeds (e.g., Vizlas and Dachshunds).

The long-coated breeds start with skin lesions and hair loss on the head, ear flaps, muzzle, and along the back +/- belly. Standard Poodles often present with symmetrical, partial alopecia and mild hyperkeratosis (crusting). There is dull/brittle hair that easily epilates, easily becomes matted, and is covered with silvery scales. The Akita will often present at an age >5yrs when compared to poodles (late onset). Ear infections are often a primary signs. The skin may have generalized red (erythematous), bumps (papules), pustules (pimples), and scales (greasy keratosebaceous debris). There may be generalized/partial alopecia. Rarely, fever and weight loss are reported. The Samoyed has similar signs as the Standard Poodle. The Belgian Sheepdog may have ear problems or even be photoaggrivated. German Shepherds may have lesions that start on the tail and progress forward (cranially).

The short-coated breeds usually present with multi-focal to coalescing patches of hair loss ("moth-eaten") or even nodular lesions without the classical scaling and crusting. The Vizslas usually has multi-focal (moth-eaten), circular or diffuse alopecia with some mild scaling (fine, white, non-adherent) +/- intermittent swelling of muzzle, lips, and eyelids. This breed (and others) may also present with coalescing nodular lesions and plaques. Dachshunds may have a similar presentation.

German Shorthaired Pointers: Exfoliative Cutaneous Lupus Erythematosus (ECLE)

- AKA: Hereditary Lupoid Dermatosis of German Shorthair Pointers
- An exfoliative form of cutaneous lupus (Olivry)
- Target: Basal Keratinocytes
- Now thought to be auto-immune; hereditary suspected (littermates can be affected)

Clinical signs

- Exfoliative dermatitis, scales/crusts
- First appear on head/back +/- generalized
- Biopsy: interface dermatitis, satellitosis**,** lymphocyte exocytosis**,** lack sebaceous glands (sebaceous adenitis)

Diagnosis

Sebaceous adenitis is diagnosed based on a clinical index of suspicion, history, and confirmatory biopsies. Other conditions must also be excluded.

Clinical management

Sebaceous adenitis is primarily a cosmetic disease. All modes of therapy (topical and oral) are intended to slow the progression of the disease and manage the skin condition.

Oral therapies have included vitamin A (10,000-20,000 IU twice daily), synthetic retinoids (isotretinoin 2-3 mg/kg/day – inhibitory effects of keratinocyte proliferation), tetracycline + niacinamide, cyclosporine (immuno-suppressive properties and inhibitory effects of keratinocyte proliferation), omega 3 fatty acids, and oral antimicrobials (antibiotics and/or anti-fungals) as needed. Topical therapies include shampoos and sprays

meant to control/inhibit the scaling/flaky skin and to help restore lubricants to the skin (sulfur + salicylic acid; propylene glycol, lactic acid, urea, phytosphingosine, oils, etc.).

Prognosis

The prognosis is usually good, but is based upon the response to therapy. Sebaceous adenitis is primarily a cosmetic disorder (affecting the appearance), but there is a possibility for chronic infections, fevers, and weight loss (e.g., some Akitas).

Sebaceous adenitis is often sub-clinical for numerous years or a patients' entire life, thus enabling the genes to be passed on to future generations.

Vitamin A responsive dermatitis (VARD)

Background

Vitamin A (retinol) is a fat-soluble vitamin that is essential for differentiation and maintenance of skin (epithelial tissue).

Dogs are able to convert plant sources of vitamin A (carotenes) in their diet to retinol (form necessary for dogs). Vitamin A Responsive Dermatosis (VARD) is a rare primary keratinization disorder.

Deficiency of vitamin A may lead to thickening and crusting of skin (hyperkeratosis). There may also be changes (squamous metaplasia) of a variety of epithelial surfaces (i.e., hyperkeratosis of sebaceous glands—blocking secretion).

There can also be abnormal cornification (process of building normal skin) and desquamation (process of ridding/shedding dead skin). There may also be diminished wound healing. Findings are compatible with phrynoderma seen in humans, which is a disease thought to be associated with vitamin A deficiency

Cause and pathogenesis

This skin disease in dogs is NOT caused by an absolute dietary deficiency of vitamin A, but more likely due to a localized abnormality in the uptake or utilization of Vitamin A by the skin. This condition is believed to be hereditary (mode of inheritance is unknown). There is an over representation of this condition in American Cocker Spaniels, Labrador Retrievers, Miniature Schnauzers, and Gordon Setters.

Clinical signs

There may be localized or generalized red bumps (papular eruptions) with firm centers, hair loss (alopecia), excessive dandruff (scaling), and multi-focal raised firm (plaque-like) crusted lesions (hyperkeratotic plaques). There may also be an increased susceptibility to microbial infection (yeast and bacterial infections). Lesions tend to be worst on the ventral thorax and abdomen/belly region.

These animals often have recurrent ear infections. The hair coat may be dry, dull and unkempt. There is often follicular plugging (debris adherent to the hairs). Gordon Setters have an atypical presentation in that they present very itchy and usually have red bumps/dermatitis over the back (dorsum).

Diagnosis

Diagnosis is achieved based on the clinical presentation, physical examination, history, biopsy results, and response to therapy. Other skin conditions that present with similar signs must be ruled-out (i.e., bacterial infection, auto-immune disease, other cornification/keratinization disorders, etc.).

Treatment

Vitamin A (10,000 IU – 20,000 IU once to twice a day) supplementation is the treatment of choice. Side-effects are unlikely, but normal tear production and occasional blood work is monitored. Clinical improvement is usually noticed within 1-3 months.

The secondary infections must be addressed as well as other potential concurrent skin problems. Lifelong maintenance therapy is usually required. The doses used for therapy are in excess of the normal dietary requirement for vitamin A in dogs.

Hypothyroidism

This is a condition in which the thyroid gland is not producing enough thyroid hormone (diminished circulating levels of thyroxine).

This is the most common endocrinopathy diagnosed in dogs.

The clinical signs for hypothyroidism often include recurrent ear infections, bilaterally symmetrical hair loss (primary and secondary hairs- dull, dry, brittle hair coat), secondary bacterial infections, weight gain, and lethargy.

Other clinical signs include hypothermia (cool seeking), personality alterations, and swelling of the face ("tragic face") due to alterations of the material that makes-up the deep skin layer (dermis/collagen/mucin).

This is most commonly seen in dogs between 6-10 years of age or earlier in large/giant breed dogs. If the results indicate that this is hypothyroidism, we will start supplementation with levothyroxine.

Recheck the bloodwork 4-6 weeks after starting therapy for 4-6 hour post pill T4 level. It is very important that the test be in that time frame.

Cutaneous T-Cell Lymphoma (CTCL)

Background

Epitheliotrophic (affecting skin) lymphoma is a malignant neoplasm (cancer) that arises from a certain population of cells in the body's immune system. The cells that are usually affected are T lymphocytes. The T-lymphocytes infiltrate the epidermis (skin) and follicular outer root sheath (hair follicle). This is an uncommon skin disease that affects dogs and cats. This disease can be generalized or multifocal. In at least 30% of the cases, the lymph nodes are involved.

Who is at risk?

Usually animals affected are older. In dogs, Scottish terriers, Golden Retrievers, English Cocker Spaniels, Gordon Setters, Cardigan Welsh Corgis, Portugese Water Dogs, Border Collies, Bulldog (English), Shar-Pei dogs, and many others are predisposed or overrepresented. In cats, like dogs, the cause is unknown and cats tend to be feline leukemia virus negative (some reports indicate positive cats are predisposed). Females are slightly more at risk for the disease and the average age is 9-11 years old.

Pathogenesis

The pathogenesis is largely unknown, but believed to be associated with chronic antigen (inflammatory stimulation), which increases skin homing T lymphocytes. There are complex immunological pathways that likely play a role. Sometimes special tests (immunohistochemistry) allow one to determine the exact cell population(s) involved. There are several stages to this disease, including the: (1) erythematous/exfoliative/erythroderma stage (red and scaling skin); (2) plaque/nodular; (3) mucocutaneous (i.e., oral lesions); (4) ulcerative (oral only). Traditionally, this disease progresses as follows: patch stage (rare), then plaque stage (solitary or multiple red plaques), than tumor/nodule stage.

Clinical signs

Dogs may present with red (erythematous) lesions accompanied by alopecia and extensive epidermal scaling, ulcerative lesions, which tend to be circular and extend through the deeper layer of skin (dermis) into the subcutis (under the skin). Cutaneous symptoms may include single to multiple plaques and/or nodules, which range from a few millimeters to several centimeters in diameter. Mucocutaneous depigmentation (mouth, nose, feet, around the vulva) and ulceration, and/or generalized erythema, alopecia, excessive scaling (dandruff), and pruritus (itching) may occur. ulcerative stomatitis (oral lesions) may be present. Most cases occur as a slowly progressive disease, and with chronicity peripheral lymphadenomegaly and signs of systemic involvement may be seen. Sometimes the footpads may be thickened (hyperkeratotic), ulcerated, or depigmented. Multiple subcutaneous swellings may be setected. Systemic symptoms include weight loss, anorexia or diarrhea. There may be blood and bone marrow changes. Sometimes there are enlarged lymph nodes.

Diagnosis

History and clinical presentation are important for providing clues that the skin disease may be cutaneous lymphoma. Cells microscopically appear abnormal (abundant round neoplastic lymphoid cells which are often histiocytic, with basophilic cytoplasm and pleomorphic, indented to lobular nuclei). Histopathology (biopsies) reveal classical findings (lichenoid band of pleomorphic neoplastic lymphocytes which infiltrate the superficial dermis and surface follicular and sweat gland epithelia. Neoplastic cells may occur within small intraepidermal vesicles (Pautrier's microabcesses).

Affected animals should be screened for internal metastasis. In addition to the skin biopsies, other diagnostic tests include a complete blood cell count, chemistry screen, x-rays, ultrasound, lymph node aspirates, and bone marrow samplings. Not all tests need to be performed, and except for the last test, these are all non-invasive

Treatments

Usually treatments tried depend on the severity of the disease and what an owner wishes to try. The goal of therapy is to not make the treatment worse than the disease. Comfort and quality of life are the most important principles. Usually a combination of chemotherapeutic medications are used, such as prednisone (corticosteroid), alkylating agents (i.e., lomustine, chlorambucil, cyclophosphamide), and others (L-asparaginase, vincristine). Synthetic vitamin A (retinoids, such as isotretinoin) may improve clinical signs in some affected animals. Some have reported a 50% clinical improvement. This may be costly, especially if the newer retinoids such as bexarotene are used. In theory, the retinoids causes cell death (apoptosis) of the cutaneous T-lymphocytes) and they downregulate certain receptors involved.

The most common treatments used currently include a combination chemotherapy protocol of prednisone, CCNU (lomustine), and L-asparaginase. These medications all have potential side effects, including increased drinking, urinating, appetite, panting, and blood panel changes. In addition, supplementation with safflower oil (which contains high levels of linoleic acid) may improve clinical signs in some animals. Immunomodulatory therapy is also tried, such as interferon. Management by a veterinary oncologist may be indicated.

Prognosis

If there are no systemic effects, the lesions may spontaneously regress in 6 months to a year (not very common). The prognosis largely depends on the stage (number of organ systems involved) and also response to initial therapy. Regardless of treatment, prognosis is usually poor, with most animals surviving less than 12 months after diagnosis. However, one must keep in mind that many affected animals are already older.

Zinc responsive dermatosis

Background

- skin contains approximately 20% of the total body zinc stores (epidermis containing 6 times more than the dermis)
- maintenance of normal keratogenesis
- classified as two distinct syndromes
- mild hyperkeratotic dermatitis, usually responds rapidly to oral zinc supplementation.

Syndrome I: Background: Siberian Huskies and Alaskan Malamutes (poor zinc absorption) +/- other (e.g., Boston Terriers); breed predilection strongly supports a genetic linkage; increased risk if: dogs on high-calcium or high-cereal diets (high levels phylates), show poor zinc absorption & high iron levels (well water, old pipes) may interfere with zinc absorption or prolonged enteritis and diarrhea; Siberian Huskies- associated possibly with hypothyroidism & decreased serum zinc level skin lesions develop despite well-balanced diets with sufficient zinc; develop early in adulthood (1 to 3 years of age) and progress at a variable rate (onset can range from 6 months to 10.5 years); 41% of dogs developing lesions before 2 years old (but many patients develop the signs later in life); Clinical Signs: >50% have lesional pruritus/itching (pruritus in "normal" skin can be the hallmark of a pending relapse); dull hair coat erythema → alopecia → crusting → scaling and underlying suppuration +/- hyperpigmentation face primarily affected: around the mouth, chin, eyes, and ears margins (+/- scrotum, prepuce, vulva, etc.) unilateral initially → symmetrical as the disease progresses; thick crusts (elbows and other pressure points) footpads may become hyperkeratotic and claws may be affected: onychomalacia clinical signs;may be precipitated or intensified by stress, illness, and estrus +/- decreased sense of smell (hyposmia) and taste (hypogeusia) may be evident.

Syndrome II: Background: Occurs in rapidly growing puppies or young adult dogs that are fed zinc deficient diets or diets high in phytates or certain minerals;Great Danes, Doberman Pinschers, Beagles, German Shepherds, German Shorthaired Pointers, Labradors, Rhodesian Ridgebacks and Standard Poodles have been reported; some animals may be normal, others stunted, depressed, +/- anorectic, +/- pyrexia +/- lymphadenopathy;

footpads primarily affected; hyperkeratotic plaques over areas of repeated trauma, footpads and nasal planum; thickened areas may have deep fissures; +/- secondary infection of the crusts and an associated lymphadenopathy

- Boston terrier – unique presentation

Ear margin seborrhea

- Primary keratinization disorder that is often asymptomatic
- Certain breeds are predisposed (Dachshund**, Cocker Spaniels & Springer Spaniels); possible variant in Boxers and Pit Bulls (at the apex)
- Affects other breeds with pendulous ears.
- The cause is unknown, but possible increased risk with exposure to forced hot air or stove.
- Clinical signs include numerous small, greasy plugs adhering to the skin & hairs of the medial and lateral margins of pinnae; hyperkeratosis; chronic cases- fissuring/painful.
- The diagnosis is based on history, physical examination, and clinical index of suspicion +/- biopsy.
- Differentials include: auto-immune, ectoparasitic disease, ischemic, zinc responsive, dermatophyte, pinnal alopecia, proliferative thrombovascular necrosis, frostbite, other.
- Treatments include: sulfur-salicylic acid +/- propylene glycol +/- urea (KeraSolv®), Benzoyl peroxide shampoos and gels (Oxydex®), Vitamin A or Retinoids (oral and topical), Pentoxifylline (oral) +/- Omega 3 Fatty Acids.

Cutaneous adverse drug reactions

Background and etiology

- 1-2% of the population(rare)- immunologic reaction→ immediate reaction or several days after starting the medication
- Numerous oral antibiotics associated: penicillin, sulfa medications, and cephalosporins (often a 20% chance of cross-over between cephalosporins and penicillins)
- Topical medications (e.g., neomycin)
- Oral thyroid and oral/injectable non-steroidal anti-inflammatory (e.g., carprofen or deracoxib) medications (e.g., facial pruritus)
- Local reactions (e.g., rabies injection- ischemic dermatopathy)
- Clinical Signs
- Hair loss, crusting/exfoliative/scaling dermatitis, ulcerated/erosive skin lesions, and other findings; reactions: focal (e.g., fixed) or generalized and may occur in the mouth as well
- Diagnosis
- Clinical signs, history, physical examination, improvement when suspected agent stopped, biopsy (virtually any pattern)
- Differential Diagnosis
- Demodicosis, dermatophytosis, juvenile cellulitis, exfoliative dermatitis, auto-immune disease
- Treatment
- Remove underlying cause
- +/- Corticosteroids
- +/- Pentoxifylline
- Prognosis
- Depends on severity and response to management

Other Conditions (less common or described in other notes) with exfoliative dermatitis will be presented.
Topical treatment options: see topical therapy lecture/notes