Clinical Approach to Scaling Dermatitis

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What is scale ?

Dry Scale = seborrhea sicca = accumulations of mature epidermal keratatinocytes (corneocytes) = orthokeratosis and/or immature epidermal keratinocytes (parakeratosis)
+/-
Hair follicle keratinocytes (principally external and internal root sheaths)
+/-
Excess sebaceous secretions (seborrhea oleosa)

Crust and scale are NOT the same.
Crust = dried exudate with blood and/or leucocytes

A cause-based approach to scaling disorders

The list of differentials is very long.
Consider groupings as:

1. **Excessive production of the stratum corneum**
2. **Defects of epidermal hydro-lipid film**
3. **Ineffective exfoliation of the stratum corneum.**

Excessive production of the stratum corneum

- Congenital defects (Primary seborrhea, comedone syndrome)
- Reaction to physical agents (rubbing , pressure , UV radiation , irritant chemicals, trauma.) Non inflammatory alopecia + UV
- Infection (bacteria and dermatophytes and yeast)
- Ectoparasites (Fleas, *Sarcoptes*, *Demodex*, *Cheyletiella*)
- Reactions to disease within the epidermis - Pyoderma , pemphigus foliaceus/erythematosus. (Usually with pustules and collarettes)
- Disease of the dermo-epidermal junction (immune meditated disease, epitheliotropic lymphoma)
- Metabolic causes - Zinc responsive dermatitis, hypothyroidism, hepato-cutaneous syndrome, vitamin A responsive dermatosis, feline thymoma, poor diet
- Idiopathic keratinization defects (e.g. ear margin dermatosis, naso/digital hyperkeratosis, feline acne)

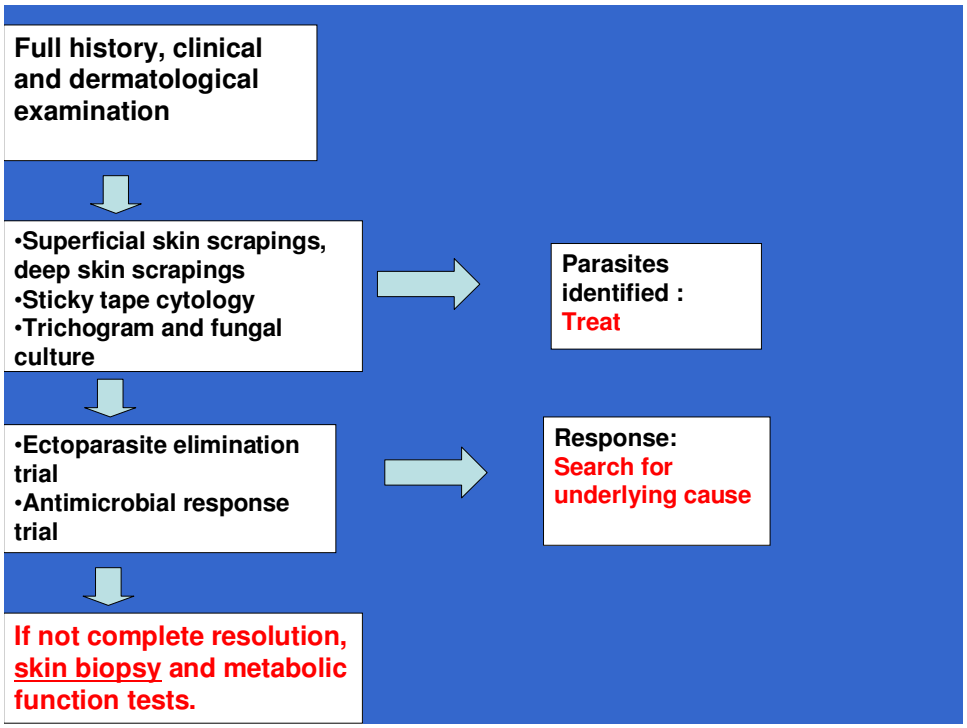
Insufficient or defective destruction and exfoliation of the stratum corneum

- Congenital ichthyosis
- Lack of grooming in sick cats

Protective surface lipid film defects

- Defects of production
  - Lack of essential fatty acids (from poor quality diets or improper storage and oxidation).
  - Destruction of the sebaceous glands (sebaceous adenitis, Leishmania)
  - Hypothyroidism
- Excessive shampoos that remove surface lipids

Clinical Approach



Symptomatic management of scale

1. Shampoo therapy
2. Hydration of skin
3. Systemic therapy
4. Control of secondary infection

Shampoos

Properties of shampoos

- Detergent (Dissolve lipids/sebum)
- Keratolytic (Break cohesion between the corneocytes)
- Keratoplastic ("Normalize" the turnover of the epidermis and the process of keratinization due to their effects on the basal epidermal cells.)
- Antibacterial and anti-Malassezia
- Moisturizing

Malaseb ®

- Good detergent action
- Antibacterial and antifungal

Selenium sulfide 1%

- Keratolytic and keratoplastic with degreasing action
- Also causes excess drying. Needs to be combined with hydration of skin
- Useful for initial therapy of heavy or greasy scale or in cases refractory to less aggressive therapy.

Coal Tar products

- Very aggressive keratolytic and keratoplastic
- Variable contents of tar components
- No longer registered for Veterinary use

Benzyl Peroxide 2-4%

- Disinfectant, astringent and keratolytic properties. Pyoben ® Virbac = 3%.
- Reputed capacity to flush debris from hair follicles.

- Good degreasing action
- Causes excessive drying of the skin. Hydrating conditioner is required after each bath.
- Dark coated dogs tend to bleach or discolour. Do not use in cats. Toxic if ingested.
- Generally used short term

Hydration of the Skin

- **Hygroscopic (humectant) agents** are high molecular weight molecules that hydrate the epidermis osmotically via dermal vasculature. Examples include urea, glycerin, lactic acid and propylene glycol. Propylene glycol has antiseptic, lipid solvent and keratolytic properties. The author often prescribes a spray using 25-33% propylene glycol in water for hydration or 45-70% as a keratolytic.

- **Emollients** , such as paraffin oil, decrease epidermal water loss. Used as a post-bath conditioner after the skin has been hydrated.

Systemic therapy

Retinoids

- Act directly on nuclear receptors
- "Normalize" keratinocyte and differentiation.
- Benefits are not evident until 2-3 months of therapy.
- Dose can be tapered down for maintenance.
- Potential side effects of retinoid therapy include keratoconjunctivitis sicca, liver damage and alterations in lipid metabolism.
- Highly teratogenic, and should not be used in breeding animals. Effects may extend for years after administration has ceased. Clients need to be informed of the teratogenic potential of these agents and should wear gloves during administration.

Vitamin A (600-800 IU/kg daily) is less potent than synthetic retinoids, acting on cytoplasmic receptors and then on the nucleus via transport molecules.

Fatty acid supplements improve dry scale. Linoleic acid, as found in sunflower or safflower oil, decreases scale and improve coat quality. No specific dose rates have

been clinically trialed, but doses of 1ml /2-4 kg are indicative. Excessive use can result in diarrhea. Omega3/6 fatty acid supplements (1ml/4kg daily) have an anti-inflammatory effect. Cold pressed products recommended

Golden Retriever Ichthyosis

Pathogenesis

- **Inherited. Autosomal recessive or incomplete dominance**
- **Recognized clinically but only recently characterized**
- **Primary keratinization defect with electron-microscopic membrane lesions in strata granulosum and corneum and retention of desmosomes between mature keratinocytes.**

Clinical signs

- May present from 12 weeks – 4 years of age.
- Flakes of dry scale from small to 1cm3 +/- focal areas of flakey seborrhea oleosa.
- Scale accumulation in ears +/- otitis externa
- Not pruritic unless secondary bacterial or yeast infection present

Differential diagnosis

- Allergic dermatitis (pruritic/inflammatory)
- Demodicosis and other skin parasites
- Primary or secondary pyoderma /Malassezia infection
- Hypothyroidism
- Definitive diagnosis is histological after secondary infection has been resolved.

Therapy

- Shampoo therapy. Selenium sulfide initially (if severe) then maintenance with sulfur/salicylic acid shampoo weekly.
- Vitamin A therapy
- Linoleic acid supplementation
- Control of secondary infections

Nasal Hyperkeratosis

1. Hereditary nasal parakeratosis of Labrador retrievers and crosses.

2. Nasal Hyperkeratosis Associated with Xeromycteria "Parasympathetic nose”

3. Senile idiopathic naso/digital hyperkeratosis.

Hereditary nasal parakeratosis of Labrador retrievers and crosses.

- Autosomal recessive
- Lesions usually appear before 12 months of age.
- Adherent accumulations of dry scale. In more severe cases, fissures and erosions develop.
- Secodary infection and inflamation may lead to depigmentation of the remaining nasal planum.
- Not exacerbated by UV light exposure.
- Sometimes clinically seen in other breeds

Nasal Hyperkeratosis Associated with Xeromycteria "Parasympathetic nose”

- The lateral nasal gland of the dog is responsible for the moisture of the nasal mucosa. Duct opens 2cm from nares and moisture translocates over the surface of the planum.
- Parasympathetic innervation (together with lacrimal glands) with the facial nerve.
- May be injured in otitis media
- Lesions may be unilateral or bilateral.
- Resolution of otitis media (if present) may result in spontaneous improvement.

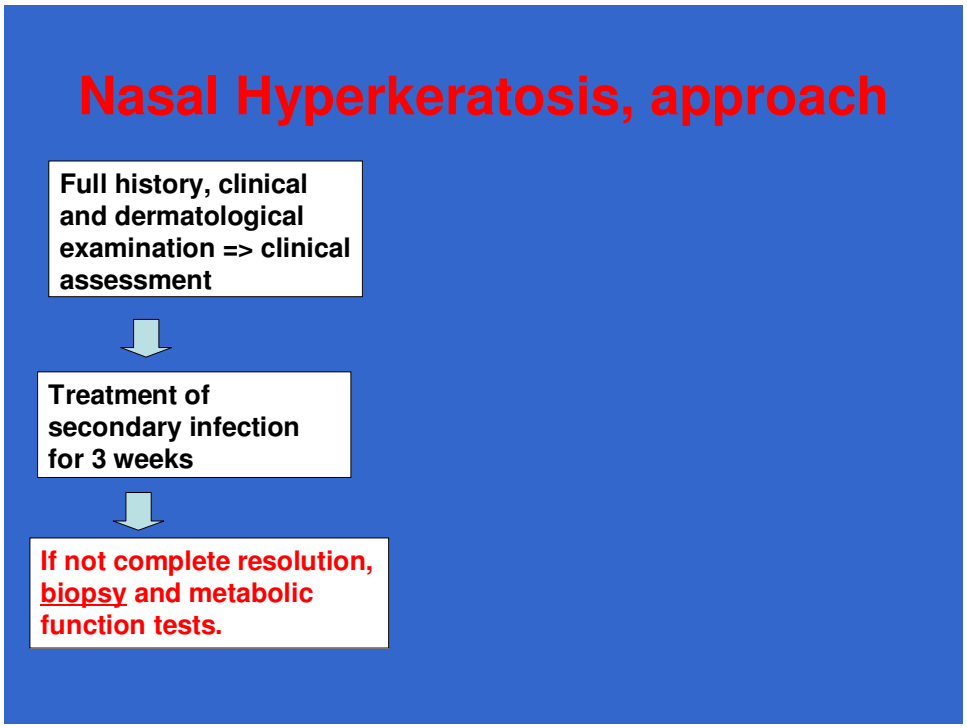
Senile idiopathic naso/digital hyperkeratosis

- Older dogs

- Increased horny tissue on the nose and/or footpads accompanied in some cases by fissures and secondary infection.
- Projections of firm, feathered, and cracked horny tissue. In some cases only the margins of the nose or footpads are affected while in others, the entire nose or footpad area can be affected.

Differential diagnosis

- Immune mediated disease
- Mucocutaneous pyoderma
- Hepatocutaneous syndrome
- Zinc responsive dermatosis
- Post distemper hyperkeratosis (hardpad)



Therapy

- **Needs to be tailored to individual.**
- Water soaks and petroleum jelly
- 50-60% propylene glycol
- Glycerin or 10% urea humectants
- Topical and systemic antibiotics for secondary infection
- Systemic fatty acid therapy
- Vitamin A?
- Topical corticosteroids??

Hepatocutaneous Syndrome (superficial epidermal necrolysis, necrolytic migratory erythema)

Pathogenesis and presentation

- Degeneration and hyperplasia of keratinocytes associated with
- chronic liver disease
- diabetes mellitus
- Pancreatic glucagonoma (less commonly in dogs (vs humans)
- Aetiopathogenisis unclear (defective protein and/or zinc metabolism?)
- Usually presents in older dogs.
- Skin signs may present before signs of liver disease

Clinical signs = Necrotizing dermatitis with hyperkeratosis

- Footpad hyperkeratosis +/- fissures
- Erythema and erosion with hyperkeratosis
  - Mucocutaneous junctions (lips, nasal planum and genitalia)
  - Friction points (elbows, hocks and distal extremities
  -

Variable laboratory findings

- Elevated liver enzymes
- Decreased albumen
- Elevated post-prandial serum bile acids
- Hyperglycemia
- Non-regenerative anemia.
- Ultrasound may reveal changes to the hepatic parenchyma that are more severe than suggested by biochemistry.

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Differential Diagnosis

- Zinc responsive dermatitis
- Immune mediated disease
- Mucocutaneous pyoderma
- Naso-digital hyperkeratosis

Definitive diagnosis is histopathology of affected areas without secondary infection and confirmation of underlying hepato/pancreatic disease.

Treatment

- Prognosis is often poor unless underlying disease can be corrected.
- Suggested therapies
  - Amino acid supplements.
    - Body builder amino acid supplements (xylatol free)
    - Chicken stock
    - Eggs,
    - Amino acid infusions

    - Zinc supplements
    - Moisturizing hyperkeratosis areas
    - Essential fatty acids
- Treatment of secondary bacterial and yeast infections
- Corticosteroids????

Sebaceous Adenitis

Pathogenesis

- Inflammation (granulomatous) and destruction of the sebaceous glands
- Probably immune mediated (cell mediated) with an inherited predisposition

Sebaceous glands functions:

- Holocrine glands: slough into central duct
- Maintains pliability of skin and hair by lubrication and retaining moisture
- Production of antimicrobial lipids for the surface hydro-lipid film

Presentation

- Presents in early to middle age
- Predisposed breeds: Akita, Standard Poodle, Springer Spaniel, Nordic breeds, Samoyed, Hovawart, Visla but any other breed can be affected

Long-haired Breeds

- Accumulations of adherent scale and follicular casts.
- Inflammatory alopecia
- Pruritus associated with secondary infection
- Poodles begins on head. Other breeds more multifocal
- Usually accompanied by cerumenous otitis

Short-haired Breeds

- Circular or fingerprint-like areas of alopecia, often beginning on head that coalesce.
- Very fine scale and +/- very fine follicular casts

Differential diagnosis

- Zinc responsive dermatosis (Nordic breeds)
- Pyoderma/folliculitis (especially Nordic breeds)
- Demodicosis and other skin parasites
- Allergic dermatitis
- Dermatophytosis
- All other listed causes of scale

Definitive diagnosis is based on skin biopsy and histopathology

Treatment Objectives

- Restore the hydration and barrier function of the skin
- Arrest destruction of sebaceous glands and permit regeneration
- Removal of scale

- Treat and prevent secondary bacterial and yeast infection

Topical therapy protocol

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Step 1: - Lathering the dog with a combined product of sulfur and salicylic acid.

- Leaving the shampoo on for a minimum of 10 minutes
- Gentle brush-massage during this time which helps to remove a significant amount of scales
- Thorough rinse-off and towel dry

Step 2: - Oil soak with any light mineral oil containing bath oil (generic baby oil).

- This is rubbed in the hair coat and allowed to soak for two hours.

Step 3: - Removal of the oil by a final lathering with a mild cleansing and antimicrobial shampoo

Step 4: - Final application of a conditioner or a mixture of propylene glycol and water (final concentration of 50-70% propylene glycol), which acts as a humectant. This mixture can be additionally applied in the times between the more labour intensive oil soaks.

Systemic therapy of sebaceous adenitis

Cyclosporine

Cyclosporine is a fungal-derived polypeptide that acts by inhibiting the enzyme calcineurin. It has a variety of immunological effects on multiple members of the skin immune system, acting by reducing the expression of and response to multiple cytokines. In particular, the inhibition of interleukin-2 production results in the apoptosis rather than activation and proliferation of T helper lymphocytes.

Clinical protocol

- 5mg/kg SID.
- **Multiple reports of efficiency** provided not all of sebaceous gland structures totally destroyed.
- Systemic drug of choice but off label
- In case of gastrointestinal side effects, the following protocol has been used:
  - Give half dose for the first five to seven days.
  - Give cyclosporine with food, half an hour after metoclopramide.
  - Discontinue the metoclopramide on day 10
  - After day 14, give on an empty stomach to maximize absorption

Ketoconazole and cyclosporine

- The metabolism and excretion of cyclosporine is an active process. Pumping of the drug through the cell membrane via microsomal cytochrome P-450. Ketoconazole actively competes for cytochrome P-450, increasing blood levels of cyclosporine.
- The effect of ketoconazole on cyclosporine blood levels is dose dependent. Ketoconazole at 4.7mg/kg SID has been shown to reduce cyclosporine dose rates needed for steady blood levels by average of 38% while doses of 13.6mg/kg SID reduced cyclosporine doses by 75%. There is considerable individual variation..
- Clear clinical protocols using ketoconazole and other agents to reduce cost are yet to be defined but many authors recommend the use of ketoconazole at 10mg/kg SID, given concurrently with or just before cyclosporine, 2mg/kg (indicative dose).
- The use of ketoconazole in the off-label manner has benefits in reducing Malassezia colonization but patients should be monitored for adverse effects, especially hepatitis.

Retinoids - Poodles and Vislas may respond better to isotretinoin while the Akita seems to benefit more from acitretin.

Vitamin A - dubious

Essential fatty acid therapy

- Omega 3- anti-inflammatory
- Omega 6 Linoleic acid – restoring barrier

Appropriate topical +/- systemic antimicrobial therapy