**Clinical Approach to Scaling Dermatitis.**

**OUTLINE**

- What is scale?
- Huge differential list. A cause-based clinical approach
- Symptomatic therapy
- Examples:
  - Golden retriever ichthyosis
  - Idiopathic nasal hyperkeratosis
  - Hepatocutaneous syndrome
  - Sebaceous adenitis

**What is scale?**

- **Dry Scale** = seborrhea sicca = accumulations of mature epidermal keratinocytes (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.
Dry Scale (Seborrhea sicca)
- Hypothyroidism
- Demodicosis

Seborrhea oleosa
- Congenital & nevi
- Malassezia
- Bacterial overgrowth syndrome
- Male feminization syndrome

Alopecia and dry scale on the flank secondary to exposure from endocrine alopecia.

Follicular Casts
- Dry Scale adhering to hair shafts
- Severe follicular pathology
Follicular Casts and dry scale

Comedones

- Commonly associated with Cushing’s disease or demodicosis.
- Indicate severe follicular disease
- Keratin-sebum plugs in hair follicles.

A cause-based approach to scaling disorders

The list of differentials is very long.

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

(1) Excessive production of the stratum corneum
- Congenital defects (Primary seborrhea, Nevi)
- Reaction to physical agents (UV radiation, irritant chemicals, trauma.)
- Infection (bacteria and dermatophytes and yeast)
- Ectoparasites (Fleas, Sarcoptes, Demodex, Cheyletiella)
- Reactions to disease within the epidermis or dermo-epidermal junction - Pyoderma, immune-mediated disease, 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)
(2) Insufficient or defective destruction and exfoliation of the stratum corneum

- Congenital ichthyosis
- Lack of grooming in sick cats

(3) 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

### Idiopathic keratosis/Nevi

- Idiopathic keratosis
- Epidermal dysplasia

### Scaling with collarettes and crusts.

**DDX** Pyoderma or Pemphigus Foliaceus

### Scale as a reaction to pyoderma

- Especially Nordic breeds
- **DDX** includes
  - Zinc responsive dermatosis
  - Malassezia
  - Sebaceous adenitis
  - Pemphigus
Scaling dermatitis due to disease at dermo-epidermal junction

Lupoid dermatosis of German Short-haired Pointers

Scaling form of pemphigus foliaceus.
Lesions not limited to head so ↔ P. erythematosus

Don’t forget: Sarcopes, Demodex and Dermatophytes

Dermatophytosis trichogram: “rotten log appearance”
Symptomatic management of scale

- Shampoo therapy
- Hydration of skin
- Systemic therapy
- Control of secondary infection

Full history, clinical and dermatological examination
- Superficial skin scrapings, deep skin scrapings
- Sticky tape cytology
- Trichogram and fungal culture
- Ectoparasite elimination trial
- Antimicrobial response trial

Parasites identified: Treat
Response: Search for underlying cause

If not complete resolution, skin biopsy and metabolic function tests.

Symptomatic management of scale
General 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

Sulfur and Salicylic acid
- Keratolytic & keratoplastic
- Synergistic actions at 2% concentration
- Moderate antimicrobial action
- Generally tolerated by cats
- Not aggressively drying
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

- Disinfectant, astringent and keratolytic properties.
- Reputed capacity to flush debris from hair follicles.
- Good degreasing action
- Causes progressive 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 agents (humectants)
  - High molecular weight molecules that hydrate the epidermis osmotically via dermal vasculature.
  - Urea, glycerin, lactic acid and propylene glycol.
  - Propylene glycol has antiseptic, lipid solvent and keratolytic properties.
  - 25-33% propylene glycol in water for hydration or 45-70% as a keratolytic

Hydration of the Skin

- Emollients
  - Decrease epidermal water loss.
  - E.G. paraffin oil
  - 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.
- Expensive, cost may limit their use. Acitretin 1mg/kg

Other systemic agents

- 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.
- Vitamin D3 (calcitriol 10ngm/kg daily) has off label retinoid-like action but has the potential of causing severe disturbances in calcium/phosphorous metabolism. Monitoring is required.
- 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.

Golden Retriever Ichthyosis

- 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.

Golden Retriever Ichthyosis Clinical signs

- May present from 12 weeks – 4 years of age.
- Flakes of dry scale from small to 1cm² +/- focal areas of flakey seborrhea oleosa.
- Scale accumulation in ears +/- otitis externa
- Not pruritic unless secondary bacterial or yeast infection present.
Golden Retriever Ichthyosis

Typical scale

Focal greasy scale

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.

Management

• Shampoo therapy. Selenium sulfide initially (if severe) -> maintenance with sulfur/salicylic acid shampoo weekly.
• Vitamin A
• Linoleic acid supplementation
• Control of secondary infections
Idiopathic Nasal Hyperkeratosis
- Hereditary nasal parakeratosis of Labrador retrievers and crosses.
- Nasal Hyperkeratosis Associated with Xeromycteria “Parasympathetic nose”
- 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.
- Secondary infection and inflammation may lead to depigmentation of the remaining nasal planum.
- Not exacerbated by UV light exposure.
- Sometimes clinically seen in other breeds

Inherited nasal hyperkeratosis

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.

Nasal Hyperkeratosis, diagnosis

- Immune mediated disease
- Mucocutaneous pyoderma
- Solar dermatitis
- Hepatocutaneous syndrome
- Zinc responsive dermatosis
- Neoplasia
- Post distemper hyperkeratosis

Nasal Hyperkeratosis, approach

Full history, clinical and dermatological examination => clinical assessment

Treatment of secondary infection for 3 weeks

If not complete resolution, biopsy and metabolic function tests.

Idiopathic nasal hyperkeratosis, Therapy

- Needs to be tailored to individual.
- Water soaks and sorbolene or petroleum jelly
- 50-60% propylene glycol
- Topical and systemic antibiotics for secondary infection
- Systemic fatty acid therapy
- Vitamin A?
- Topical corticosteroids??
Hepatocutaneous syndrome
(superficial epidermal necrolysis, necrolytic migratory erythema)

- Degeneration and hyperplasia of keratinocytes associated with:
  - Chronic liver disease
  - Diabetes mellitus
  - Pancreatic glucagonoma. Less common in dogs vs humans
- Aetiology/pathogenesis unclear (defective protein and/or zinc metabolism?)
- Older dogs
- Skin signs may present before signs of liver disease

Hepatocutaneous Syndrome

Necrotizing dermatitis + hyperkeratosis

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

Laboratory findings

Variable

- 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.

Biochemistry values in hepatocutaneous syndrome

Table 1. Biochemistry results in dogs with hepatocutaneous syndrome

<table>
<thead>
<tr>
<th>Biochemical parameter</th>
<th>Case 1</th>
<th>Case 2</th>
<th>Case 3</th>
<th>Case 4</th>
<th>Case 5</th>
<th>Reference values</th>
</tr>
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<tbody>
<tr>
<td>Total protein (g/dL)</td>
<td>5.9</td>
<td>6.1</td>
<td>6.2</td>
<td>6.3</td>
<td>6.4</td>
<td>5.2-7.0</td>
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<tr>
<td>Albumin (g/dL)</td>
<td>3.7</td>
<td>3.9</td>
<td>3.8</td>
<td>3.9</td>
<td>4.0</td>
<td>2.7-4.5</td>
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<td>Glucose (mg/dL)</td>
<td>115</td>
<td>112</td>
<td>114</td>
<td>116</td>
<td>118</td>
<td>60-140</td>
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<tr>
<td>Hematocrit (%)</td>
<td>41.3</td>
<td>41.5</td>
<td>41.2</td>
<td>41.4</td>
<td>41.6</td>
<td>38-45</td>
</tr>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>12.5</td>
<td>12.3</td>
<td>12.4</td>
<td>12.3</td>
<td>12.5</td>
<td>13.0-16.0</td>
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<tr>
<td>Total bilirubin (mg/dL)</td>
<td>0.6</td>
<td>0.5</td>
<td>0.6</td>
<td>0.5</td>
<td>0.6</td>
<td>0.2-1.4</td>
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<tr>
<td>Direct bilirubin (mg/dL)</td>
<td>0.3</td>
<td>0.2</td>
<td>0.3</td>
<td>0.2</td>
<td>0.3</td>
<td>0.1-0.4</td>
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<tr>
<td>Aspartate aminotransferase (U/L)</td>
<td>25</td>
<td>23</td>
<td>24</td>
<td>22</td>
<td>23</td>
<td>15-45</td>
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<tr>
<td>Alanine aminotransferase (U/L)</td>
<td>20</td>
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<td>17</td>
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<td>10-35</td>
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<tr>
<td>Alkaline phosphatase (U/L)</td>
<td>95</td>
<td>90</td>
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<td>91</td>
<td>92</td>
<td>40-130</td>
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<tr>
<td>Creatinine (mg/dL)</td>
<td>1.2</td>
<td>1.1</td>
<td>1.2</td>
<td>1.1</td>
<td>1.2</td>
<td>0.7-2.0</td>
</tr>
</tbody>
</table>

Hepatocutaneous syndrome

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

Definitive diagnosis:
- Histopathology (without secondary infection)
- Demonstration of underlying cause
Hepatocutaneous syndrome

- Prognosis is often poor unless underlying disease can be corrected.
- Suggested therapies:
  - Amino acid supplements (e.g. eggs, infusions?)
  - Zinc supplements (ZnSO$_4$, 10mg/kg)
  - Moisturizing hyperkeratosis areas
  - Essential fatty acids
- Treatment of secondary bacterial and yeast infections
- Corticosteroids?

Sebaceous Adenitis

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

Sebaceous glands

Clinical aspects

- 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

Clinical signs

- 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
- 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
Early stage of sebaceous adenitis in an Akita. Tightly adhering scale and secondary infection

Advanced stage of sebaceous adenitis in an Akita. Tightly adhering scale, follicular casts, otitis and secondary infection

Sebaceous adenitis 3yo Maltese

Appearance of sebaceous adenitis in short-haired breeds.
**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 of sebaceous adenitis**

**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**

- **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**

- **Cyclosporine.**
  - 5mg/kg SID. **Multiple reports of efficiency** provided not all of sebaceous gland structures totally destroyed.

- Systemic drug of choice but off label
**Cyclosporine mechanisms**

- The metabolism and excretion 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.

**Ketoconazole and cyclosporine**

- Other systemic therapies for sebaceous adenitis
  - Retinoids - $$$ and teratogenicity
  - Vitamin A
  - Essential fatty acid therapy
    - Omega 3/6 - anti-inflammatory
    - Linoleic acid – restoring barrier
  - (Pulse?) antimicrobial therapy

**Thank you**

Questions and discussion

References available on request