4.5 DERMATOLOGY – SKIN CONDITIONS OF PRIMATES

C. Colin and W. Boardman

Skin diseases are common in primates. They are often seen in new arrivals at sanctuaries, or they can flare up in groups because of the contagious nature of many skin conditions. Some are zoonoses, so they can be transmitted to the staff, or the staff can transmit them to the primates as well! Skin disorders are often can be a sign of or secondary to a generalised problem. For example, immunocompromised individuals, either from another disease, or secondary to stress (e.g. newcomers, dominancy issues or other behavioural issue in a group), will often show evidence of a skin disorder.

It’s quite hard to detect primary skin lesions because they are too ephemeral to be observed, but they are really important for the diagnostic, as they are the direct reflect of the disease. So, when looking for skin lesions, take care and look cautiously, several times per day, or day after day to have a chance to find and identify primary lesions.

When dealing with skin disorders, prevention is better than cure so attempt to:

- manage new primate arrivals to your sanctuary to prevent potential spread of any skin condition being carried, (See section 1.2)
- have a good quarantine protocol, to prevent the new primate from transmitting any potential diseases to the rest of the primates in the sanctuary and to the staff (see section 1.2 and 3.4)
- have effective cleaning procedures of facilities and staff hygiene (see Section 1.2 and PASA Operations Manual)
- reduce the stress within the animals in the sanctuary as much as possible, including the risk of diseases transmission among the groups (refer to the PASA Operations Manual and the first 2 sections of this manual).

1. GENERAL EXAMINATION

Before concentrating on dermatologic examinations, start with a general examination, to see if there are other health problems, related to the skin manifestations:

- TºPR
- Mucosa: colour, lesions
- Examine every system (respiratory, cardiac, digestive, urinary, musculo-skeletal, nervous)

2. DERMATOLOGIC EXAMINATION

Several goals:

- To find cutaneous lesions
- To identify the lesions
- Localization and distribution of the lesions
- Check for ectoparasites
2.1 DESCRIPTION OF THE LESIONS: There are 3 kinds of skin lesions – primary, secondary or mixed. Skin can only show disease in a set number of ways. By correctly describing the lesions, you will have a better chance of arriving at the correct diagnosis quickly, and providing appropriate therapy

**PRIMARY LESIONS:** they are a direct reflection of a skin disease, they are ephemeral. They are really important to make a correct diagnosis

<table>
<thead>
<tr>
<th>With colour change</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Macule</strong></td>
<td>small, flat, circular spot, color changed (vitiligo, viral exanthem, drug eruptions) Several macules = <strong>patch</strong></td>
</tr>
<tr>
<td><strong>Purpura</strong></td>
<td>red or purple discolorations due to erythrocytes extravasations</td>
</tr>
<tr>
<td><strong>Petechia</strong></td>
<td>Punctiform lesion</td>
</tr>
<tr>
<td><strong>Echymoses</strong></td>
<td>Large spots</td>
</tr>
<tr>
<td><strong>Erythema</strong></td>
<td>redness of the skin due to capillary congestion (sign of inflammation)</td>
</tr>
</tbody>
</table>

**The ‘solid’ lesions**

<table>
<thead>
<tr>
<th><strong>Papule</strong></th>
<th>solid raised lesion. Several = <strong>plaque</strong>. Seen in allergic reaction (=wheal); Bacterial, fungal or parasitic infections for follicular papules; atopy or scabies for non follicular papules</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Nodule</strong></td>
<td>solid lesion more deeply rooted than a papule. Lipomas, fibromas, etc</td>
</tr>
<tr>
<td><strong>Tumor</strong></td>
<td>bigger, abnormal growth or mass of tissue</td>
</tr>
<tr>
<td>----------</td>
<td>------------------------------------------</td>
</tr>
<tr>
<td><strong>Cyst</strong></td>
<td>cavity delimited by epithelium, that may contain air, liquid or semi/solid material</td>
</tr>
<tr>
<td><strong>Full of liquid</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Vesicle</strong></td>
<td>raised lesion full of clear liquid. Large one = <strong>bullae</strong>. <a href="#">Readily breakable lesions.</a> Insect bites, characteristic lesion of Herpes infection</td>
</tr>
</tbody>
</table>
| **Pustule** | raised lesion filled with pus, results from an infection  
  - Follicular pustules: bacterial, fungal folliculitis  
  - Non follicular pustules: impetigo |

**THE SECONDARY LESIONS:** they result from a natural evolution of the primary lesions. They can be the result of a fungal or bacterial superinfection, or a consequence of scratching or treatment. These lesions are the most frequently observed, but they have little diagnostic interest

<table>
<thead>
<tr>
<th><strong>With abnormal production</strong></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Epidermal collarette</strong></td>
<td>more or less circular edge, formed by some or totality of the epidermis, which remains after the bursting of a lesion I (vesicle, pustule)</td>
</tr>
<tr>
<td><strong>Crust</strong></td>
<td>dried collection of blood, serum or pus, consequence of rupture of lesion filled with liquid. Healing process</td>
</tr>
</tbody>
</table>

<p>| <strong>With lost of substance</strong> |  |</p>
<table>
<thead>
<tr>
<th>Lesion</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Erosion</td>
<td>loss of epidermis</td>
</tr>
<tr>
<td>Excoriation</td>
<td>erosion with traumatic origin, e.g. result of scratching</td>
</tr>
<tr>
<td>Sclerosis</td>
<td>indurations of the skin</td>
</tr>
<tr>
<td>Atrophy</td>
<td>rarefaction of dermis components, giving the skin a thin and wrinkled appearance</td>
</tr>
<tr>
<td>Lichenification</td>
<td>skin thickening, often with hyper-pigmentation, resulting from inflammatory process</td>
</tr>
</tbody>
</table>

**THE MIX LESIONS:** they can be Primary or secondary.

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ulcer</td>
<td>deep loss of substance (dermis or hypodermis). <strong>Fissure</strong> if really deep</td>
</tr>
<tr>
<td>Alopecia</td>
<td>reduction of hairs density or lack of hairs</td>
</tr>
<tr>
<td>Scale</td>
<td>dry, gray or whitish build-up of dead skin cells</td>
</tr>
</tbody>
</table>
Keratoseborrhetic state  dysfunction of cutaneous lipid production associated with keratinization troubles. Can be greasy or dry (e.g. ectoparasites)

2.2 TECHNIQUES OF SKIN EXAMINATION

Skin scrape:
- Basic and easy examination
- Provide information from epidermis, dermis
- To determine presence/absence of ectoparasites, fungus in epidermis, in hair follicles
- For detection of mites, make several ones, in areas with papules/crust with a scalpel blade blunted
- Put collected material on a slide with lactophenol, examine under 10× as a scan, then at 40× to increase chance of correct diagnosis of any findings

Swab:
- In cases of otitis (ear infection), swabs can be used to collect material from the external auditory canal
- Put collected material on a slide
  - With lactophenol for Otodectes and other mites
  - Roll the swab on a slide, staining to look for inflammatory cells and Malassezia spp. (fungus - yeasts)

Combing of the hair coat:
- To collect skin debris and cutaneous parasites including lice, fleas, ticks, some mites, etc.
- Collect these into a container or on a clean surface (see section 3.17 for storage)

Scotch (Cellotape) test:
- To detect ectoparasites. This is also used around the anus to detect pinworm.
- Paste the piece of scotch on a slide, and examine directly under 10× for detection of lice, 40× for dermatophytisis
- With staining for detection of Malassezia sp. under 100×

Cytology if available:
- To identify bacteria, fungi, inflammatory or neoplastic cells
- Impression smears on exsudative or ulcerating lesions
- Rupture pustules and vesicles with a sterile needle
- Stain slides and examine under 40× and 100× (oil immersion).

Others if available: biopsy, bacterial and fungal cultures, routine blood and urine tests, etc.
3. **SKIN DISEASES**

<table>
<thead>
<tr>
<th>INFECTIOUS</th>
<th>NON-INFECTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ectoparasites</strong></td>
<td><strong>Nutritional disorders</strong></td>
</tr>
<tr>
<td>Scabies mite, lice, fleas, chigger, tumbu fly</td>
<td><strong>Behavioral disorders</strong></td>
</tr>
<tr>
<td><strong>Bacteria</strong></td>
<td><strong>Toxic</strong></td>
</tr>
<tr>
<td>Impetigo, folliculitis, cellulitis</td>
<td><strong>Fungi</strong></td>
</tr>
<tr>
<td><strong>Fungi</strong></td>
<td><strong>Allergic</strong></td>
</tr>
<tr>
<td>Ringworm, candidiasis</td>
<td><strong>Virus</strong></td>
</tr>
<tr>
<td><strong>Virus</strong></td>
<td><strong>Neoplastic</strong></td>
</tr>
<tr>
<td>Filoviruses, Herpes viruses, Pox viruses, Papilloma viruses</td>
<td></td>
</tr>
</tbody>
</table>

### 3.1 ECTOPARASITES

<table>
<thead>
<tr>
<th>Class</th>
<th>Ordre</th>
<th>Family</th>
</tr>
</thead>
<tbody>
<tr>
<td>Scabies mite</td>
<td>Arachnida</td>
<td>Acarina</td>
</tr>
<tr>
<td>Ticks</td>
<td>Arachnida</td>
<td>Acarina</td>
</tr>
<tr>
<td>Chiggers</td>
<td>Arachnida</td>
<td>Acarina</td>
</tr>
<tr>
<td>Lice</td>
<td>Insecta</td>
<td>Anoplura</td>
</tr>
<tr>
<td>Fleas</td>
<td>Insecta</td>
<td>Siphonaptera</td>
</tr>
<tr>
<td>Tumbu flies</td>
<td>Insecta</td>
<td>Dipterae</td>
</tr>
</tbody>
</table>

**Scabies**
- Caused by mites, *Sarcoptes scabei*
- Characterized by superficial burrows, intense pruritus (EXTREMELY itchy) and secondary infections
- Spreads easily within a group, by skin to skin contact, holding facilities (indirect transmission)
- Potential zoonosis
- Adult female mite burrows into the outside layers of the skin
- Lays up to 3 eggs/day during her lifetime of 1-2 months.
- The development from egg to adult requires 10 to 14 days
- Scabies burrows hard to notice on skin of non-human primates
- Rash with itchy nodules (allergic reaction to the mites)
- Loss of hair, thickening and wrinkling of the skin and scab and crust formation.
- Secondary infection common, because of intense scratching
- Hyperkeratosis in extreme cases
- Can affect all the body
Symmetrical lesions

Hyper keratosis

Ticks
- Can transmit a lot of pathogens
- Crimean-Congo Hemorrhagic Fever (CCHF virus, fam. Bunyaviridae), a tick-born virus transmitted by *Hyalonema sp.* to wild and domestic animals, humans
- Rift Valley Fever (RVF virus, fam. Bunyaviridae) transmitted by *Aedes sp* but also by ticks.

Chiggers = Harvest mites (genus *Trombicula*; also known as red bugs, trombiculid mites, scrub-itch mites, berry bugs or, in their larval stage, as chiggers)
- Larvae feed on skin cells
- Post larval stage are not parasitic
- Responsible for severe itching, with papules and skin rashes, possible secondary infection due to scratching
- Do not spread from host to host
- Do not carry disease in Africa

Fleas
- *Pulex irritans*: human flea
- *Ctenocephalides felis* and *P. irritans* found on monkeys
- Some fleas are really host specific, but most of them can feed on numerous hosts
- Can be vector of major zoonoses (plague, typhus)
- *Bartonella quintana* found in *P. irritans* on a pet monkey (*Cercopithecus cephus*) in Gabon
- Monkeys can be infected by *Rickettsia* and be a reservoir as well as humans
Tumbu fly
- *Cordylobia anthropophaga*: "Tumbu fly" or "ver de Cayor" or "mango fly" or "skin maggot fly."
- Furuncular myasis due to larva of fly
- Eggs laid on the ground on dry, sandy soil often contaminated with urine and feces of animals. Eggs hatch after 1-3 days, larvae can wait up to 15 days for a host. Skin penetration really quick
- Evolution of 1-2 weeks, larva then emerges through the breathing pore and falls to the ground
- Small erythematous papule 2-3 days after larval penetration
- Evolution into a nodule which looks like a boil, with a pore in the center (where the larva breathes)
- Remove larvae manually (coating the breathing pore with a thick, viscous compound helps, e.g. vaseline)
- Surgery if necessary
- Great care when removing larva, rupturing larva might cause anaphylactic reaction
- Antibiotics recommended

### Treatment

<table>
<thead>
<tr>
<th></th>
<th>Local treatment</th>
<th>General treatment</th>
<th>Treatment of environment</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Scabies mite</strong></td>
<td>Ascabiol, Spregal</td>
<td>Ivermectine 200µg/kg</td>
<td>Good disinfection of resting and sleeping areas</td>
</tr>
<tr>
<td></td>
<td>2 or 3*/10 or 14days</td>
<td>(up to 5 days) antibiotics</td>
<td></td>
</tr>
<tr>
<td><strong>Ticks</strong></td>
<td>Manual removal</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Chiggers</strong></td>
<td>Hot bath with soap</td>
<td>(Anti-Hi, antibiotics)</td>
<td></td>
</tr>
<tr>
<td><strong>Lice</strong></td>
<td>Permethrin 1% 2*/7days</td>
<td>Ivermectine 200µg/kg</td>
<td>Clean</td>
</tr>
<tr>
<td><strong>Fleas</strong></td>
<td>Pet treatments?</td>
<td></td>
<td>Pyriproxifen or Methoprene repeated</td>
</tr>
<tr>
<td><strong>Tumbu flies</strong></td>
<td>Manual removal</td>
<td>Antibiotics</td>
<td></td>
</tr>
</tbody>
</table>


3.2 BACTERIAL INFECTIONS

The natural defenses of the skin are composed of cutaneous endogenous flora, which are not primarily pathogenic, the physico-chemical properties of the skin and immunologic factors (immunoglobulins are secreted in sweat from Langerhans cells). **Inducing factors** to a bacterial skin infection, include breakdown in one or more of these barriers due to poor hygiene, a skin alteration, a local or general corticosteroid therapy, or use of other immunosuppressive drugs, or anything that can cause an immunodepression. In many of cases, *Staphylococcus spp.* is implicated.

<table>
<thead>
<tr>
<th>Bacteria</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Impetigo</td>
<td>group A beta-hemolytic <em>Streptococcus</em> (GABHS), and/or <em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>Folliculitis</td>
<td><em>Staphylococcus aureus</em></td>
</tr>
<tr>
<td>Cellulitis</td>
<td><em>Staphylococcus aureus</em>, <em>S. pyogenes</em>, in association with <em>Streptococcus</em> (and others)</td>
</tr>
</tbody>
</table>

**Streptococcal infections = IMPETIGO:**
- Highly contagious gram+ infection of the superficial layers of the epidermis.
- Due to group A beta-hemolytic *Streptococcus* (GABHS), and/or *Staphylococcus aureus* (most of the time association of both)
- Can be a primary infection or a surinfection
- 2 kinds : bullous and non bullous
  - **Bullous form**: Rapid onset of blisters that enlarge and rupture
  - **Non-bullous form**: Starts with a single erythematous macule which evolves rapidly into a vesicle or pustule, ruptures leaving a crusted yellow exudate over the erosion

**Staphylococci infections**
- **Superficial folliculitis:**
  - Eruption of small pustules, each one around a hair
  - Do not confuse with dermatophytosis folliculitis

- **Deep folliculitis (furunculosis):**
  - Acute infection of a hair follicle due to *Staphylococcus aureus*
  - Area can be hard, red, hot, painful
  - Fever and adenopathy possible

- **Cellulitis :**
  - Acute infection of the skin and soft tissue characterized by localized pain, swelling, tenderness, erythema, warmth
  - Caused by *Staphylococcus aureus* and *S. pyogenes*, but association with *Streptococcus*, and other bacteria!
  - Always a primary lesion (trauma, insect bite, or systemic disease like diabetes)
Other symptoms: fever, chills, myalgia, etc.

Treatment

<table>
<thead>
<tr>
<th>Local</th>
<th>General</th>
<th>Remark</th>
</tr>
</thead>
<tbody>
<tr>
<td>Impetigo</td>
<td>Antibiotic creams 2-3 times/day for 7-10 days</td>
<td>Cephalosporines, semi-synthetic penicillin, If no improvement/resistance: tetracyclines, Bactrim©, clindamycin</td>
</tr>
<tr>
<td>Antiseptic, antibiotic creams</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Folliculitis</td>
<td></td>
<td>General treatment if bad</td>
</tr>
<tr>
<td>Cellulitis</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3.3 FUNGAL INFECTIONS:

Ringworm
- Dermatophytes due to 3 genera: *Trichophyton*, *Microsporum* and *Epidermophyton*
- A large variety of dermatophytes (*T. rubrum* most common in the world)
- Zoonotic infections
- Environment, people, animals are carriers
- Can stay in environment: recurrent outbreaks can occur
- Direct and indirect transmissions
- Dermatophytes are in the cornified layers of the skin.
- Incubation period of 1-3 weeks
- Self-limited infection in healthy individuals but can widespread in young or debilitated animals
- Increased epidermal cell proliferation in response to infection, with resultant scaling.
- *Tinea corporis:* first lesions are eythematous, scaly plaques, and then formation of annular shape. Crusts, vesicles, papules often develop.
  - Not always itchy in animals
  - Dry lesions, red and circular, really typical
- Tinea corporis in human and in a chimpanzee

Candidiasis
  - Yeast, *Candida albicans*
  - Superficial skin infections and mucous membrane
  - A commensal fungus of gastro-intestinal tract of humans
  - Affects immuno-depressed patients
  - Induced by trauma, skin maceration, endocrine diseases, nutritional deficiencies (Fe, Vit B1, B2, B6, folic acid), low immune system
  - Cutaneous forms in humans but NHP?
  - Oropharyngeal candidiasis OPC = oral thrush, confirmed in chimpanzees:
    - OPC may clear spontaneously
    - Common in immuno-depressed infants
    - Pearly white patches on mucosal surface
    - Lesions may progress to symptomatic erosions and ulcerations
    - Can become systemic, but rare

Treatment

<table>
<thead>
<tr>
<th>Local</th>
<th>Systemic</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Ringworm</strong></td>
<td>Topical azoles (<em>econazole, ketoconazole, miconazole, sertaconazole</em>, etc.) on the area and 2cm around, twice a day at least for 2 weeks</td>
<td><strong>Griseofulvin</strong>: 10 to 15mg/kg/day for 6 weeks <strong>Ketoconazol</strong>: 3-4mg/kg/day but risk of hepatitis <strong>Fluconazole</strong>: 1mg/kg BID for 2 weeks</td>
</tr>
<tr>
<td><strong>Candidiasis</strong></td>
<td>Nystatin Myconazole Clotrimazole topical cream</td>
<td>Fluconazole (Diflucan®) PO: <strong>Adults</strong>: 200mg PO single dose or 100mg/day for 5 days (for thrush) <strong>Infants</strong>: 3-6mg/kg/day 14-28 days</td>
</tr>
</tbody>
</table>
3.4 VIRAL SKIN INFECTIONS

Viruses are important pathogens in tropical areas; and many of them have muco-cutaneous manifestations. Viruses can spread quickly, mutate and cross species barriers. We now face the emergence of new viruses in Africa due to population growth and natural habitat destruction. Both Apes and humans can be efficient vectors for these viruses!

FILOVIRUSES
- Ebola, Marburg viruses, responsible for severe hemorrhagic fevers.
- Ebola kills gorillas, chimpanzees and humans (and other wild animals)
- People can get infected by manipulating infected dead apes, meat, blood, etc.
- **In humans, characteristic, non puritic, maculopapular centripetal rash associated with erythema, which desquamate by day 5-7 of the illness.**
- Hemorrhagic manifestations develop at the peak of the illness, with ecchymoses, disseminated petechiae on skin and mucous membranes
- **Rash is characteristic in humans affection, for differential diagnostic**

HERPES VIRUSES
- *Herpes virus hominis*: HSV1, HSV2, EBV, CMV, VZV, etc
- **Zoonotic**
- Herpes B (*Herpesvirus simiae*), enzootic in rhesus, cynomolagus and other OWM of Macaca genus
- Like *Herpes hominis*, Herpes B characterized by lifelong infection with intermittent reactivations. Transmission among group through sexual activities and bites
- **Can be zoonotic, few human cases, but really severe (lethal in most of the cases)**
- In humans, vesicular skin lesions at or near site of inoculation + localized neurological symptoms, and at the end encephalitis
- In primates, no or mild symptoms

Simian Varicella-Zoster virus (SVV)
- Closely related to Varicella-Zoster virus (VZV), which causes varicella only on humans
- Induces a varicella-like disease in OWM: fever and vesicular skin rash on the face, torso and the extremities (green and patas monkeys, also chimps and gorillas)
- Can produce a latent infection in neural ganglia and reactivate
- High morbidity and mortality
- Direct transmission by inhalation or direct skin to skin contacts
POXVIRUSES

- **Orthopoxviruses**:
  - Smallpox only on humans (NHP experimental)
  - MONKEYPOX (humans, NHP, rodents)
    - Orthopoxvirus with enzootic circulation in rain forests of central and western Africa
    - Zoonose, transmission by direct contact with an infected animal, indirect by eating undercooked meat, inoculation from cutaneous or mucosal lesions of an infected animal especially with compromised skin barrier (bites, scratches, other…)
  - *In NHP (in captivity)*:
    - Multiple and discrete papules (1-4 mm diameter)
    - *Lesions abundant in the palms of the hands, whole trunk and tail*
    - Content of papules is very thick, similar to pus. Sometimes ulcerative circular lesions in the mouth.

- **Parapoxviruses**

- **Yatapoxviruses**: Tanapox virus and Yabapoxviruses (humans and NHP):
  - **Tanapox virus**
    - Human cases in Kenya in the 1950’s, and in the Sates in personnel of primate centers working with macaques
    - Nothing known about pathology in wild primates but studies showed that *Cercopithecus aethiops* could be a reservoir
    - In humans, skin lesions (nodules – papules – ulcers, then healing process)
    - In NHP in captivity, enlarged circles of skin with umbilication and adherent scab center. Lesions found primarily on the face (around the lips and nostrils) but also on other parts of the body.
    - Transmission by arthropods
  - **Yabapox virus**:
    - 1st case known in Yaba, Nigeria, in 1958 in a *Cynomolgus* colony
    - Causes subcutaneous tumor-like growth in monkeys
    - Lesions on the extremities, less frequently on the face.
    - Spontaneous regression of the lesions

- **Papilloma viruses**:
  - Responsible for warts
  - PV replicate on the skin or mucosal surfaces (mouth, genitals, anus, airways), in the keratinocytes
  - Transmission by direct contact via micro-lesions of the skin
  - Some PV lead to cancer but not frequent
3.3 NON INFECTIOUS SKIN DISEASES

a. Nutritional
Nutritional deficiencies are common in new arrivals at a sanctuary (see section 3.7, Malnutrition). In cases of malnourished animals (especially protein and vitamin deficiencies) you can see edema of the face, loss of muscles, associated with hair loss, thin and rough dry hair, pigment loss, associated or not with ectoparasites, ringworm, bacterial infections of the skin… and other clinical signs (intestinal malabsorption, leaver disease, etc.).
You must be careful with group living animals as there are always problems of dominance and of food access. Be sure that every single animal receive enough food, in quantity and quality at every meal. The food quality is a really important factor (See nutrition section 3.6 and its recommended texts).

b. Behavioral
This can be seen in different contexts in a sanctuary:
- Case of a new arrival: stress of previous captivity, of changes, new group…
- Stress among the group, bad dynamic in the group. Improvements are usually spectacular if you move the animal in another group where he will feel more at ease
- Auto mutilation behaviors in extreme cases of psychologic trauma
- Over self grooming if an animal get bored or is stressed. This can be improve with a change of group or by improving enrichment of the living conditions

c. Allergic reactions to plants (by contact or ingestion), caterpillars, insects…in the environment
- Intense pruritus with big papules. Usually, disappears in few hours
- Anti-Hi, corticoids by injection if it is really bad or on young animals
- Be careful with generalized allergic reaction, edema of the face and the throat = emergency = Quick edema (corticoids, epinephrine injectable). See Section 3.8 (Emergency Medicine)

d. Toxic
- Snake bite: pain, swollen, bleed, blister, necrosis at the bite site. General symptoms, like internal hemorrhages, nervous symptoms, muscle death, etc.
- Cleaning products: good use / kept in a safe place
- Cleaning protocol with the staff to avoid cleaning in presence of animals
Topical treatments for ectoparasites, rinse carefully!

e. Neoplastic: it is really rare.

References


Emerging infectious diseases www.cdc.gov/edi V11 (11), November 2005

Demarche diagnostique en dermatologie canine  Thèse de doctorat vétérinaire, A Lecourt 2005

Démarche diagnostique en dermatologie feline en vue de l’établissement d’une nouvelle fiche clinique de consultation Thèse de doctorat vétérinaire, C Toing-Poux 2005 152p

http://emedicine.medscape.com/dermatology

Devienne, P. Bobard, P. Pinhas,C Le Ver de Cayor, agent d’une myase furonculeuse


http://www.merckvetmanual.com/

http://www.pediatrics.wisc.edu/education/derm/master.html

http://www.lib.uiowa.edu/HARDIN/MD/DERMPICTURES.HTML

http://tray.dermatology.uiowa.edu/DermImag.htm

http://health.stateuniversity.com/pages/1421/Skin-Lesions.html for descriptions and drawings of skin lesions

http://www.webmd.com/
Basic Principles

- Hygiene – direct contact reduction
- Zoonoses
- Moisture reduction
- Stress reduction
- Primary skin infection Vs. manifestation of systemic disease

Manifestations of Skin Disease

- A number of disease processes can be involved in any clinical sign
- Skin only has a certain number of ways in can present with disease
- Dry, flaky skin
- Oily moist skin
- Well circumscribed or generalised lesion
- Itchy or not itchy
- Raised lesion or flat
- Break in skin or intact
- Inflammation – reddened (increased blood flow to area), hot, painful, loss of function, swelling.

Non Infectious Skin Diseases

- Nutritional – see Nutrition discussion
- Behavioural – over grooming, bites (See Wounds discussion)
- Neoplastic – Spontaneous – very rare. Some initiated by viruses
- Toxic – cleaning fluid in satellite cages
- Allergies – usually very itchy

Infectious Skin Diseases

- Fungal – Ringworm
- Viral – Pox, Papilloma, Herpes
- Bacterial – see bacterial discussion
- Parasitic – Filariasis – see parasite discussion.

Common Conditions
• Ringworm
• Dermatophytosis - zoonotic
• Fungus
• Direct contact, and via objects
• Carrier animals and in environment.
• Non-itchy, dry, red often circular lesions
• Juveniles more susceptible – if bad cases in adults – indicates underlying disease
• Associated with Vitamin A deficiency
• Treatment often not required especially if free ranging – but can last for several months.
• Miconazole (daktarin) – topical/ long term
• Griseofulvin – oral/ long term/ dangerous in females

HERPESVIRUS HOMINIS (SIMPLEX)

• Common, asymptomatic in humans
• Found in chimpanzees and gorillas
• Mild, self limiting oral vesicular lesions

MONKEYPOX VIRUS

• Original host = squirrel
• Lesions in chimps, gorillas and humans
• Direct contact
• Fever, rash.
• Human cases in Liberia, Sierra Leone, Zaire, Nigeria, Cameroon, CAR.
• Quarantine, Vaccine?

PAPILLOMAVIRUS

• Warts
• Direct contact
• Usually self limiting
• Can lead to tumours.
• Can surgically remove.

SIMIAN VARICELLA VIRUSES

• African Green Monkeys, Patas Monkeys
• Alphaherpesvirus – can be latently infected in ganglia
• High mortality/ morbidity
• Vesicles on skin – like chickenpox
• A chimpanzee and gorilla variant has been found – mild and self limiting
• More like a human form of the disease than the monkey form

SKIN MANIFESTATION OF SYSTEMIC DISEASE

e.g.
• Tip of the iceberg – gives an indication of deeper problems
• Examples only of what may see – not comprehensive
• DEEP PYODERMA
  • Pain, exudate, sloughing of skin
  • Fever, Depression, Anorexia
• FOOD HYPERSENSITIVITY
  • Chronic itchiness, papules, pustules
  • Diarrhoea
• SNAKE BITE
  • Rapid and progressive oedema, pain, sloughing of skin
  • Shock, coagulation problems, sepsis, respiratory compromise
• PROTEIN DEFICIENCY
  • Scaling, pigment loss, patchy hair loss, thin hairs, rough dry hair, delayed wound healing
  • Intestinal malabsorption, liver disease, starvation