Diseases of the Skin

The major functions of the skin are:

1- To maintain a normal body temperature
2- To maintain a normal fluid and electrolyte balance within the animal
3- To create a mechanical barrier to protect the body from noxious agents and organisms
4- To act as a sensory organ perceiving those features of the environment that are important to the subject's survival.

Primary/Secondary Lesions

Diseases of the skin may be primary or secondary in origin.

In primary skin diseasethe lesions are restricted initially to the skin, although they may subsequently spread from the skin to involve other organs. While cutaneous lesions may be secondary to disease originating in other organs.

Note: *Differentiation between primary and secondary skin diseases should be attempted by seeking evidence that organs other than the skin areaffected.*

Clinical Signs and Special Examination

A general clinical examination is followed by a special examination of the skin and must include:

A- inspection
B- palpation.
C- Additional special examination:
    1- swabs for bacteriologic examinations.

2- scrapings for examination for dermatophytes and metazoan parasites.
3- biopsy for histopathological examination. Biopsy material should include abnormal, marginal, and normal skin.
4- A Wood's lamp finds a special use in the examination of the skin for dermatophytes.
5- Descriptions of lesions should include size, depth to which they penetrate topographic distribution on the body, and size of the area affected. Abnormalities of sebaceous and sweat secretion, changes in the hair or wool coat, and alterations in color and temperature of the skin should be noted, as should the presence or absence of pain.

Lesions

Scales: Dry flaky exfoliations

Excoriations: Traumatic abrasions and scratches.

Fissures: Deep cracks.

Dry gangrene: Dry, horny, black, avascular, shield-like.

Keratosis: Overgrowth of dry, horny, keratinized epithelium.

Acanthosis: Like keratosis but moist, soft.

Hyperkeratosis: Excessive overgrowth of keratinized, epitheliumlike

Scab.

Parakeratosis: Adherent to skin.

Eczema: Erythematous, itching dermatitis

Discrete lesions

Vesicle, bleb, bulla, Blister : Fluid (serum or lymph)-filled blister 1–2 cm diameter.

Pustule: Pus-filled blister, 1–5 mm.

Wheal: Edematous, erythematous, swellings, transitory.

Papules (pimples): Elevated, inflamed, necrotic center, up to 1 cm diameter.

Nodules, nodes: Elevated, solid, up to 1 cm diameter Acute or chronic inflammation. No necrotic center.

*Plaqu*e: Larger nodule, up to 3–4 cm diameter.

Acne: Used synonymously with *pimple* but strict meaning is infection of sebaceous gland.

Impetigo: Flaccid vesicle, then pustule, then scab, up to 1 cm diameter.

Scab (or crust): Crust of coagulated, blood, pus, and skin debris.

Macule (patch): Small area of color change; patch is larger.

Abnormal Coloration

The parameter of abnormal coloration includes *jaundice, pallor, and erythema.* In animals these conditions are rarely visible in light-colored skins.

Early erythema is a common finding where more definite skin lesions are to develop, as in early photosensitization.

The blue coloration of early gangrene (e.g., of the udder and teat skin in the early stages of peracute bovine mastitis associated with *Staphylococcus aureus*) is characterized by coldness and loss of elasticity.

Pruritus

• **Pruritus or itching** is the sensation that gives rise to scratching.

• **Hyperesthesia** is increased sensitivity to normal stimuli.

• **Paresthesia** is perverted sensation, a subjective sensation, and not diagnosed in animals.

Common causes include the following.

Cattle and sheep

• Sarcoptic and chorioptic mange

• Lice infestation

• Nervous acetonemia

Horses

• Chorioptic mange on the legs

• Queensland (sweet) itch along the dorsum of the body

• Lice infestation

• Perianal pruritus from *Oxyuris equi* infestation.

Abnormalities of Wool and Hair Fibers

Deficiency of hair or wool in comparison to the normal pilosity of the skin area is **alopecia** or **hypotrichosis**.

Diseases of the Epidermis and Dermis

PITYRIASIS

Pityriasis scales are accumulations of keratinized epithelial cells, sometimes softened and made greasy by the exudation of serum or sebum.

Primary pityriasis is characterized by excessive bran-like scales on the skin and is caused by overproduction of keratinized epithelial cells. The etiology is uncertain.

The diagnosis is based on clinical presentation and can be supported by further diagnostic testing ruling out other differential diagnoses.

Proposed

causative or predisposing factors are as follows:

1- Hypovitaminosis A
2- Nutritional deficiency of B vitamins.
3- Poisoning by iodine

Secondary pityriasis is characterized by excessive desquamation of epithelial cells and usually associated with the following:

1- Scratching in flea, louse, and mange infestations
2- Keratolytic infection (e.g., with ringworm fungus).

Primary pityriasis scales are superficial, accumulate where the coat is long, and are usually associated with a dry, lusterless coat. Itching or other skin lesions are not features.

Secondary pityriasis is usually accompanied by the lesions of the primary disease.

Pityriasis is identified by the absence of parasites and fungi in skin scrapings.

DIFFERENTIAL DIAGNOSIS

1- Hyperkeratosis
2- Parakeratosis
3- Ringworm

TREATMENT

Primary treatment requires correction of the primary cause.

Supportive treatment commences with a thorough washing, followed by alternating applications of a bland emollient ointment and an alcoholic lotion. Salicylic acid is frequently incorporated into a lotion or ointment with a lanolin base.

HYPERKERATOSIS

Epithelial cells accumulate on the skin as a result of excessive keratinization of epithelial cells and intercellular bridges, interference layer of the epidermis, and hypertrophy of
the stratum corneum.

Local hyperkeratosis may be caused by the following:

1- Mechanical stress on pressure points (e.g., elbows, hocks, or brisket) when animals lie habitually on hard surfaces
2- Mechanical and/or chemical stress (e.g., **teat-end keratosis** of dairy cows that can be caused by improper milking machine settings, overmilking, improper use of teat sanitizers or cold weather)
3- Parasitism (e.g., hyperkeratotic form of sarcoptes mange of pigs and small ruminants)

Generalized hyperkeratosis may be caused by the following:

1- Poisoning with highly chlorinated naphthalene compounds
2- Chronic arsenic poisoning
3- Inherited congenital ichthyosis

4- Infection with a fungus, was recently associated with generalized hyperkeratosis in a calf and a goat kid.

The skin is dry, scaly, thicker than normal, usually corrugated, hairless, and fissured in a gridlike pattern. Secondary infection of deep fissures may occur if the area is continually wet.

Confirmation of the diagnosis is by the demonstration of the characteristically thickened stratum corneum in a biopsy section, which also serves to differentiate the condition from parakeratosis, inherited ichthyosis “Congenital and Inherited Skin Defects”).

Primary treatment depends on correction of the cause. Supportive treatment is by the application of a keratolytic agent (e.g., salicylic acid ointment).

PARAKERATOSIS

Parakeratosis, a skin condition characterized by incomplete keratinization of epithelial cells, can have various causes: Caused by

1- nonspecific chronic inflammation of cellular epidermis
2- Associated with dietary deficiency of zinc
3- Part of an inherited disease.

The initial lesion comprises edema of the prickle cell layer, dilatation of the intercellular lymphatics, and leukocyte infiltration. Imperfect keratinization of epithelial cells at the granular layer of the epidermis follows, and the horn cells produced are sticky and soft, retain their nuclei, and stick together to form large masses, which stay fixed to the underlying tissues or are shed as thick scales.

Initially the skin is reddened, followed by thickening and gray discoloration. Large, soft scales accumulate, are often held in place by hairs, and usually crack and fissure; their removal leaves a raw, red

surface. Hyperkeratosis scales are thin and dry and accompany an intact, normal skin.

The diagnosis is made based on the clinical presentation and can be confirmed by the identification of imperfect keratinization in a histopathological examination of a biopsy or a skin section at necropsy.

DIFFERENTIAL DIAGNOSIS

1- Hyperkeratosis
2- Ringworm
3- Sarcoptic mange
4- Inherited ichthyosis
5- Inherited parakeratosis of calves
6- Inherited epidermal dysplasia.

TREATMENT

Primary treatment requires correction of any nutritional deficiency (specifically, correcting zinc and preventing excessive dietary calcium content).

Supportive treatment includes

1- Removal of the crusts by the use of keratolytic agent (e.g., salicylic acid ointment) or by vigorous scrubbing with soapy water, followed by application of an astringent (e.g., white lotion paste), which must be applied frequently and for some time after the lesions have disappeared.

IMPETIGO

Impetigo is a superficial eruption of thinwalled, small vesicles, surrounded by a zone of erythema, that develop into pustules, then rupture to form scabs.

In humans, impetigo is specifically a streptococcal infection, but lesions are often invaded secondarily by *staphylococci*.

In animals the main organism found is usually a staphylococcus. The causative organism appears to gain entry through minor abrasions, with spread resulting from rupture of lesions causing contamination of surrounding skin and the development of secondary lesions.

Two specific examples of impetigo in large animals are as follows:

• **Udder impetigo** (udder acne) of cows

• **Contagious impetigo,** also known as **exudative epidermitis** (see also "Udder Impetigo") or "**greasy pig disease,**"

Confirmation of the diagnosis is by isolation of *staphylococci* from vesicular fluid.

DIFFERENTIAL DIAGNOSIS

1- Cowpox/buffalopox, in which the lesions occur almost exclusively on the teats andpass through the characteristic stages of pox
2- Pseudocowpox, in which lesions are characteristic and also restricted in occurrence to the teats
3- Ringworm.

TREATMENT

Primary treatment with antibiotic topically is usually all that is required because individual lesions heal so rapidly.

Supportive treatment is aimed at preventing the occurrence of secondary lesions and spread of the disease to other animals.

Twice-daily bathing with an efficient germicidal skin wash is usually adequate.

URTICARIA

Urticaria (hives) is a skin condition characterized by development of topical dermal edema becoming apparent as cutaneous wheals.

Horses are the most commonly affected species. In acute cases hives appear suddenly and regress within hours.

chronic cases are characterized by the continuous recurrence of new wheals on the skin for days or even months.

Urticaria can occur as localized allergic reaction only affecting parts of the skin or as part of a more severe systemic a allergic reaction.

Primary urticaria can be caused by the following:

1- Insect stings
2- Contact with stinging plants
3- Ingestion of unusual food, with the allergen, usually a protein
4- Occasionally an unusual feed item (e.g., garlic to a horse).
5- Administration of a particular drug (e.g., penicillin, streptomycin, possibly guaifenesin or other anesthetic agent)
6- Allergic reaction in cattle following vaccination for foot-and-mouth disease.
7- Death of warble fly larvae in tissue
8- Milk allergy when Jersey cows are dried off.
9- Transfusion reaction.
10-Cutaneous vasculitis (purpurea hemorrhagica).
11-Local skin trauma (dermatographism).
12- Temperature induced (heat, cold, sunlight)
13. Infection—parasitic, bacterial, fungal, viral.

Secondary urticaria occurs as part of a syndrome, such as Respiratory tract infections in horses, including strangles and the upper respiratory tract viral infections.

PATHOGENESIS

Degranulation of mast cells liberating chemical mediators of inflammation that result in the subsequent development of dermal edema is the presumed cause for the development of urticaria.

A primary dilatation of capillaries causes cutaneous erythema. Exudation from the damaged capillary walls causes local edema in the dermis, and a wheal develops.

CLINICAL FINDINGS

1- Wheals, mostly circular, well-delineated, steep-sided, easily visible elevations in the skin, appear very rapidly and often in large numbers, commencing usually on the neck but being most numerous on the body.

2- They vary from 0.5 to 5 cm in diameter, with a flat top, and are tense to the touch. There is often no itching, except with plant or insect stings, and no discontinuity of the epithelial surface, exudation, or weeping.

3- Pallor of the skin in wheals can be observed only in un pigmented skin.

4- Other allergic phenomena, including diarrhea and slight fever, may accompany the eruption.

DIFFERENTIAL DIAGNOSIS

Urticaria is manifested by a suddenappearance of a crop of cutaneous weals, sometimes accompanied by restlessness, mostly in horses, occasionally in cattle.

Identification of the etiology is also helpful in diagnosis but is often difficult, depending on a carefully taken history and examination of the environment.

The **differential diagnosis list** is limited to angioedema, but in urticaria the lesions can be palpated in the skin itself. Angioedema

TREATMENT

Acute anaphylaxis with urticaria in horses:

1- Epinephrine: 3 to 5 mL/450 kg of a 1 : 1000 solution IM or SC (can be combined with steroids).

Acute urticaria in horses:

1- Dexamethasone soluble 0.01 to 0.1 mg/kg IV or IM q24 h for 3 to 7 days.

Chronic or recurrent urticaria:

1- Prednisolone 0.25 to 1.0 mg/kg IV or PO q24 h. Reduce to 0.2 to 0.5 mg/kg q48 h.
2- Dexamethasone 0.01 to 0.02 mg/kg POq48-72 h . Further reduce dose until the lowest dose keeping the animal free of signs is determined.
3- Hydroxyzine hydrochloride 0.5 to 1.0 mg/kg IM or PO q8 h.
4- Diphenhydramine hydrochloride 0.7 to 1 mg/kg q12 h.
5- Chlorpheniramine 0.25 to 0.5 mg/kg q12 h

Dermatitis And Dermatosis

ETIOLOGY

Some of the identifiable occurrences of dermatitis in food animals and horses are as follows.

All Species

1- Mycotic dermatitis as a result of *Dermatophilus congolensis*, in horses, cattle, and sheep
2- *S. aureus*, a common finding in cases in all species, either as a sole pathogen or combined with other agents
3- Ringworm.
4- Photosensitive dermatitis
5- Chemical irritation (contact dermatitis) topically .
6- Arsenic—systemic poisoning
7- Mange mite infestation—sarcoptic,
8- psoroptic, chorioptic, demodectic mange.
9- Biting flies, especially *Culicoides* spp.; observed most commonly in horses, but also in other species
10- *Stephanofilaria* spp. Dermatitis
11- *Strongyloides (Pelodera)* spp. Dermatitis

Special Local Dermatitides

Special local dermatitides include dermatitis of the teats and udder, the bovine muzzle and coronet, and flexural seborrhea, and are dealt with under their respective headings.

PATHOGENESIS

Dermatitis is an inflammation of the deeperlayers of the skin involving the blood vessels and lymphatics. The purely cellular layers of the epidermis are involved only secondarily. The noxious agent causes

cellular damage, often to the point of necrosis, and, depending on the type of agent responsible, the resulting dermatitis varies in its manifestations.
It may be acute or chronic, suppurative, weeping, seborrheic, ulcerative, or gangrenous.

CLINICAL FINDINGS

1- Affected skin areas first show erythema and increased warmth. The subsequent stages
2- Edema of the skin and subcutaneous tissues may occur in severe cases.
3- The next stage may be the healing stage of scab formation; if the injury is more severe, there may be necrosis or even gangrene of the affected skin area.
4- Spread of infection to subcutaneous tissues may result in a diffuse ***cellulitis or phlegmonous*** lesion.
5- A distinctive suppurative lesion is usually classified as pyoderma.
6- A systemic reaction is likely to occur when the affected skin area is extensive.
7- Shock, with peripheral circulatory failure, may be present in the early stages.
8- Toxemia as a result of absorption of tissue breakdown products, or septicemia as a result of invasion.

Pemphigus

Pemphigus is an autoimmune disease of the skin occurring in mature horses, usually 5 years of age or older, as well as in foals.
Vesicles and pustules are usually very difficult to find because they progress rapidly to crusts, exfoliation, alopecia, and scaling.
There are a number of manifestations, of which pemphigus foliaceus is the most common.

Pemphigus vulgaris and bullous pemphigoid, in contrast, are rare. Pemphigus is a chronic autoimmune disease often accompanied by severe weight loss.

Differential Diagnosis

1- **Hyperhidrosis** and **anhidrosis** are dysfunctions of sweating and have no cutaneous lesion.
2- **Cutaneous neoplasm** is differentiable on histopathological examination.
3- **Epitheliogenesis imperfecta** is a congenital absence of all layers of skin.
4- **Vascular nevus** is a congenital lesion commonly referred to as a "birthmark."

Treatment

1- Primary treatment is removal of the (presumed) causative agent.
2- supportive treatment includes treatment for pruritus secondary infection, shock, toxemia, or fluid and electrolyte loss.

Photosensitization

Etiology And Epidemiology

Photosensitization is caused by exposure of tissue containing certain photoactive substances to light of specific wavelength.

Substances with the potential to accumulate in skin and get activated by solar irradiation are termed **photosensitizing substances (PSs)**.

Photosensitization differs from sunburn in that it requires the presence of a photosensitizing agent, it is triggered by exposure to a wavelength of light between 320 and 400 nm (in contrast to sunburn, which in most cases is the result of exposure to light with lower wavelength), its onset

is rapid (in contrast to the more delayed onset with sunburn), and skin lesions are considerably more severe than with sunburn.

photosensitization has been classified into four classes, as follows:

• **Type I,** primary photosensitization caused by PSs of exogenous origin (no underlying primary pathology of the organism).

Occur due to exogenous PSs can enter the organism through oral ingestion (e.g., PSs contained in feed), parenteral administration (e.g., certain drugs), or direct absorption through skin.

• **Type II,** photosensitization caused by aberrant pigment metabolism.

Occur due to porphyrins that may accumulate in an organism with disturbed heme synthesis.

• **Type III,** hepatogenous photosensitization caused by disturbed liver. Function such as livestock due to accumulate phylloerythrin .

• **Type IV,** idiopathic photosensitization that is of undetermined etiology.

PATHOGENESIS

Penetration of light rays to sensitized tissues causes local cell death and tissue edema. Irritation is intense because of the edema of the lower skin level, and loss of skin by necrosis or gangrene and sloughing is common in the terminal stages. Nervous signs may occur and are caused either by the photodynamic agent, as in buckwheat poisoning, or by liver dysfunction.

Clinical signs

1- Primary cases (skin lesion) have cutaneous signs only (erythema, edema, necrosis, gangrene of light-colored skin or mucosae exposed to sunlight).
2- Secondary cases have also signs of hepatic dysfunction (jaundice, prostration, short course, death).

3- **Systemic Signs** include shock in the early stages, as a result of extensive tissue damage. There is an increase in the pulse rate, body temperature with ataxia and weakness.
4- **Nervous Signs** including ataxia, posterior paralysis and blindness, and depression or excitement, are often observed.

Differential diagnosis

Clinical evidence of restriction of damage to white, wool-less skin on body dorsum and lateral aspects of limbs, teats, corneas, and tongue and lips.

Treatment

Primary: remove from exposure to sunlight and PS.

Supportive: treat for infection, shock, toxemia. Nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, or antihistamines can be administered parenterally and adequate doses maintained.