Diseases of the skin

Introduction:

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 act as a sensory organ perceiving those features of the environment which are important to the subject's survival.

In general, these functions are not greatly impeded by most diseases of the skin of large animals, with the exceptions of failure of the sweating mechanism, which does seriously interfere with body temperature regulation, and severe burns or other skin trauma, which may cause fatal fluid and electrolyte loss. The major effects of skin diseases in large animals are esthetic and economic.

Primary and secondary skin disease (Lesion):-

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

In primary skin disease the lesions are restricted initially to the skin although they may subsequently spread from the skin to involve other organs. On the other hand, cutaneous lesions may be **secondary** to disease originating in other organs. Differentiation between primary and secondary skin diseases should be attempted by seeking evidence that organs other than the skin are affected. If there is no such evidence produced during a complete clinical examination of the patient, it is reasonable to assume that the disease is primary.

Clinical signs and special examination

A general clinical examination is followed by a special examination of the skin and must include **inspection** and, in most cases, **palpation.** Additional information can be obtained by taking swabs for bacteriological examinations, scrapings for examination for dermatophytes and metazoan parasites, and biopsy for histopathological examination.

Biopsy material should include abnormal, marginal, and normal skin. Artifacts are common in biopsy specimens, including nonrepresentative sampling, crushing the specimen by forceps or hemostat, and inadequate fixation.

Wood's lamp finds a special use in the examination of the skin for dermatophytes. Descriptions of lesions should include size, depth to which they penetrate, geographical distribution on the body and size of the area covered.

Abnormalities of sebaceous and sweat secretion, changes in the hair or wool coat and alterations in color of the skin should be noted, as should the presence or absence of pain.

The main lesion of skin disease.

Name of lesion	Nature of lesion	Relation to skin surface	Skin surface
Scales	Dry, flaky exfoliations	On surface only, no penetration of skin	Unbroken
Excoriations	Traumatic abrasions and scratches	Penetration below surface	Variable skin surface damage depends on severity
Fissures	Deep cracks	Penetrate into subcutis	Disrupted Removed
Dry gangrene	Dry, horny, black, avascular, shield-like	Above skin, usually all layers affected	
Early, moist gangrene	Blue-black, cold, oozing serum	In plane of skin or below Above skin	Complete depth of subcutis Undamaged stratum corneum is retained
Keratosis	Overgrowth of dry, horny, keratinized epithelium	Above skin	Prickle cell layer swollen; is really part of skin
Acanthosis	Like keratosis but moist, soft	Above skin	Skin surface unbroken
Hyperkeratosis	Excessive overgrowth of keratinized, epithelium-like scab	Above skin	Cells of stratum corneum nucleated and retained; really part of skin
Para keratosis	Adherent to skin		Weeping, scabby disruption of surface

Eczema	Erythematous, itching dermatitis	Superficial layer of epidermis affected In epidermis or dermis	Unbroken
Hypermelanosis	Increased deposits of melanin, e.g. melanosis, meloderma	In epidermis or dermis	Unbroken
Hypomelanosis	Decreased deposits of melanin		
Discrete lesions Vesicle, bleb, bulla, blister	Fluid (serum or Lymph)-filled blister 1 -2 cm diameter	Above skin surface, superficial	Un broken but will slough
Pustule	Pus-filled blister, 1 -5 mm	Above, superficial	Will rupture
Wheal	Edematous, erythematous, swellings, transitory	Above, all layers affected	Undamaged
Papules (pimples)		Above surface, all layers affected	Points and ruptures
Nodules, nodes	Elevated, inflamed, necrotic center, up to 1 cm diameter	Above surface, all layers	Surface unbroken

Plaque	Elevated, solid, up to 1 cm diameter. Acute or chronic inflammation. No necrotic center	All layers affected; raised above surface	Surface un broken May point and rupture
	A larger nodule, up to 3-4 cm diameter	Above surface of skin; all layers affected	May rupture
Acne	Used synonymously with pimple but strict meaning is infection of sebaceous gland	Raised above skin	Upper layers destroyed
Comedo	Plugged (sebum, keratin) hair follicle	Raised above skin; very superficial	
Impetigo			
Scab (or crust)	Flaccid vesicle, then pustule, then scab, up to 1 cm diameter Crust of coagulated, blood, pus and skin debris	Raised above skin	Disrupted, depth varying with original lesion
Macule (patch	Small area of color change; patch is larger	Within superficial layers	Unbroken

Abnormal coloration

This parameter includes **jaundice, pallor and erythema**, and these are best seen in the **oral or vaginal mucosa or in the conjunctiva**. In animals they are rarely visible in light-colored skins.

Red-purple discoloration of the skin of septicemic.

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.

Hypopigmentation of the skin maybe general as in albino, pseudoalbino and lethal white animals. Local patches of hypopigmentation are characteristic of vitiligo and leukoderma.

Pruritus or itching: is the sensation that gives rise to scratching Relation to skin surface

Hyperesthesia is increased sensitivity to normal stimuli

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

All sensations that give rise to rubbing or scratching are therefore included with pruritus, more properly defined as scratching. Pruritus can arise from peripheral or central stimulation. When it is peripheral in origin it is a primary cutaneous sensation like heat, cold, pain and touch; it differs from pain because it is purely

epidermal. whereas pain can still be felt in areas of skin denuded of epidermis. Thus itching does not occur in the center of deep ulcerations nor in very superficial lesions, such as those of ringworm, where only the hair fibers and keratinized epithelium are involved. Itching can be elicited over the entire skin surface but is most severe at the mucocutaneous junctions. Common causes include the following.

Cattle : Sarcoptic and chorioptic mange, Aujeszky's disease, Nervous acetonemia, Lice infestation.

Sheep :Lice, mange, ked, blowfly and itch mite infestations Scrapie.

Pigs : Sarcoptic and chorioptic mange and Lice infestation.

Horses : Chorioptic mange on the legs, Queensland (sweet) itch along the dorsum of the body, Lice infestation and Perianal pruritus due to Oxyuris equi infestation.

All species

The early stages of **photosensitive dermatitis**, **Urticarial wheals** in an allergic reaction, '

Licking syndromes' such as occur in cattle on copper-deficient diets are accompanied by pica and the licking of others as well as themselves. They are examples of depraved appetites developed in response to nutritional deficiency and are not a response to pruritus. Itching of central origin derives in the main from the scratch center below the acoustic nucleus in the medulla. It may have a structural basis, as in scrapie and pseudorabies, or it may be functional in origin, as in the nervous form of acetonemia. The only lesions observed are those of a traumatic dermatitis with removal of the superficial layers to a variable depth, breakage or

removal of the hairs and a distribution of lesions in places where the animal can bite or rub easily.

Secretion abnormalities of skin glands

The activity of the **sweat glands** is controlled by the sympathetic nervous system and is for the most part a reflection of body temperature. **Excitement and pain** may cause sweating due to cerebral cortical activity. A generalized form of hyperhidrosis, apparently inherited, has been recorded in Shorthorn calves. Local areas of increased or decreased sweating may arise from peripheral nerve lesions or obstruction of Principles of treatment of diseases of the skin sweat gland ducts. A generalized anhidrosis is recorded in horses and occasionally in cattle. Excess secretion of sebum by sebaceous glands causes oiliness of the skin or seborrhea but its pathogenesis is poorly understood.

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. Hirsutism, abnormal hairiness, manifested by a long, shaggy, usually curly, coat is most common in aged ponies with adenomas of the pars inter media of the pituitary gland. The character of the fiber may also vary with variations in the internal environment. For example, in copper deficiency the crimp of fine wool fibers is lost and the wool becomes straight and 'steely'. Alternation in coat color, achromotrichia, may be generalized or segmental along the fiber.

Principles Treatment of Skin Diseases

Primary treatment

Primary treatment commences with removal of hair coat and debris to enable topical applications to come into contact with the causative agent. Accurate diagnosis of the

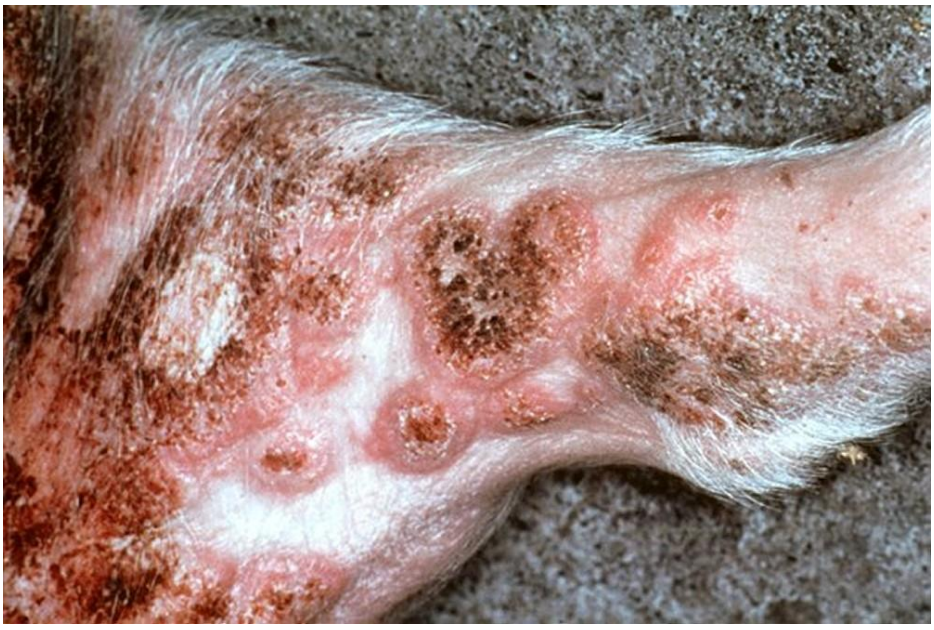
cause must precede the selection of any topical or systemic treatment. In bacterial diseases sensitivity tests on cultures of the organism are advisable. Specific skin diseases due to bacteria, fungi and metazoan parasites are reasonably amenable to treatment with the appropriate specific remedy.

Supportive treatment

Supportive treatment includes prevention of secondary infection using bacteriostatic ointments or dressings and the prevention of further damage from scratching.

- Effective treatment of pruritus depends upon the reduction of central perception of itch sensations by the use of ataractic, sedative or narcotic drugs administered systemically or on successful restraint of the mediator between the lesion and the sensory end organ. In the absence of accurate knowledge of the pathogenesis of pain it is usual to resort to local anesthetic agents, which are short lived in their activity, and corticosteroids, which are longer-acting and effective, provided that vascular engorgement is part of the pruritus-stimulating mechanism.
- When large areas of skin are involved it is important to prevent the absorption of toxic products by continuous irrigation or the application of absorptive dressings.
- Losses of fluid and electrolytes should be made good by the parenteral administration of isotonic fluids containing the necessary electrolytes Ensure an adequate dietary intake of protein, particularly sulfur-containing amino acids to facilitate the repair of skin tissues.
- Boredom contributes significantly to an animal's response to itch stimuli, and close confinement of affected animals is best avoided.

1- Pityriasis



a- Primary pityriasis,

Excessive bran-like scales on the skin, characterized by overproduction of keratinized epithelial cells, 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. pityriasis can be caused by:

1- Hypovitaminosis A
2- Nutritional deficiency of B vitamins, especially of riboflavin and nicotinic acid, in pigs, or linolenic acid, and probably other essential unsaturated fatty acids.
3- Poisoning by iodine

b- Secondary pityriasis, is usually accompanied by the lesions of the primary disease and characterized by excessive desquamation of epithelial cells is usually associated with:

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

Pityriasis scales are accumulations of keratinized epithelial cells, sometimes softened and made greasy by the exudation of serum or sebum. Overproduction, when it occurs, begins around the orifices of the hair follicles and spreads to the surrounding stratum corneum.

Diagnosis

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

Differential diagnosis

Hyperkeratosis and Parakeratosis

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.

2- Dermatitis and dermatosis

Dermatitis is a term include inflammation of dermal and epidermal tissues characterize by hyperemia, exudation, infiltration, itching and pain.

Etiology

As other skin diseases it including those characterized by inflammation, all pathogens, infectious (bacterial, viral, fungal, parasitic), chemical, physical (sun rays, cold or heat, trauma), allergic, and autoimmune.

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

Epidemiology

Sporadic or outbreak, acute or chronic course, cosmetic to lethal, but of most importance as constraints on movement, sale or exhibition

Pathogenesis

- Dermatitis is basically an inflammation of the deeper layers of the skin involving the blood vessels and lymphatics. The purely cellular layers of the epidermis are involved only secondarily. It may be acute or chronic, suppurative, weeping, seborrheic, ulcerative or gangrenous.
- In all cases there is increased thickness and increased temperature of the part. Pain or itching is present and erythema is evident in unpigmented skin. Histologically there is vasodilatation and infiltration with leukocytes and cellular necrosis. These changes are much less marked in chronic dermatitis.

Clinical signs

Primarily localized to skin, including lesions varying from parakeratosis and pachyderm a to weeping, through necrosis, vesicles and edema. Secondarily signs of shock, toxemia, anaphylaxis

Diagnosis

Case history, clinical signs, lab. Examinations (swab)

Clinical pathology

1. Examination of skin scrapings or swabs for parasitic, bacterial or other agents is essential.
2. Culture and sensitivity tests for bacteria are advisable to enable the best treatment to be selected.

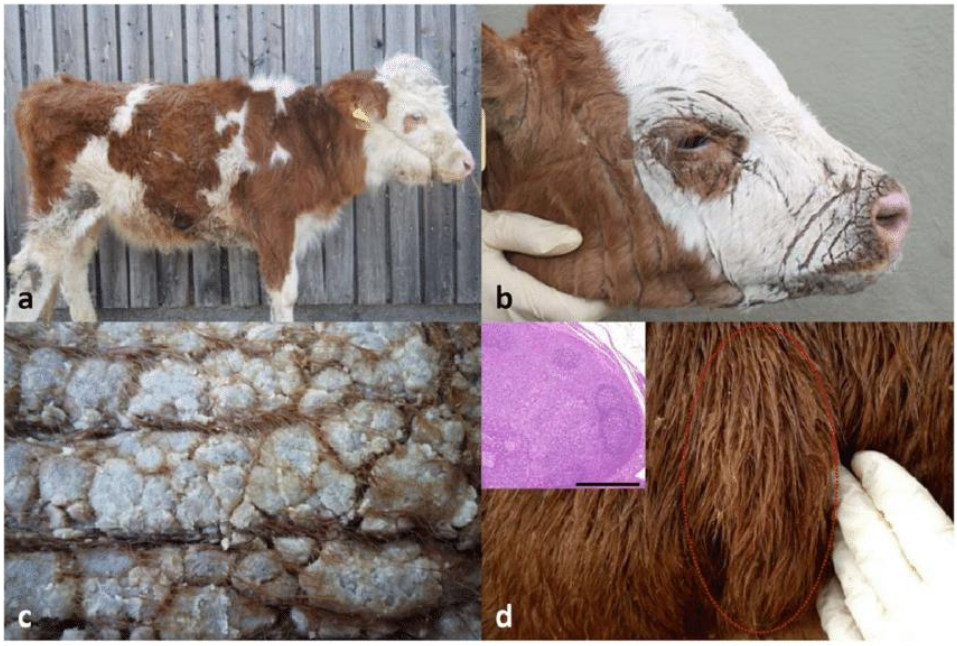
3. Skin biopsy may be of value in determining the causal agent.
4. In allergic or parasitic states there is usually an accumulation of eosinophils in the inflamed area.
5. In mycotic dermatitis organisms are usually detectable in the deep skin layers although they may not be cultivable from superficial specimens.

Necropsy lesions Inflammatory, degenerative or vascular lesions in skin biopsy

Treatment

- Primary is removal of the causative agents.
- Remove the chemical and physical surrounding the animal.
- supportive includes treatment for shock, toxemia or fluid and electrolyte loss, vitamins (A and B), proteins.
- Topical ointments and lotions.

3- Hyperkeratosis



Epithelial cells accumulate on the skin as a result of excessive keratinization of epithelial cells and intercellular bridges, interference with normal cell division in the granular layer of the epidermis and hypertrophy of the stratum corneum. It can be dividing to:

a. **local hyperkeratosis** the lesion at pressure points as elbows, when animals lie habitually on hard surfaces.
b. **Generalized hyperkeratosis** may be caused by:
   - Poisoning with highly chlorinated naphthalene compounds.
   - Chronic arsenic poisoning.
   - Inherited congenital ichthyosis.
   - Inherited dyserythropoiesis dyskeratosis.

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

continually wet. However, the lesion is usually dry and the plugs of hyperkeratotic material can be removed, leaving the underlying skin intact.

Diagnosis

By the demonstration of the characteristically thickened stratum corneum in a biopsy section.

Differential diagnosis

parakeratosis and inherited ichthyosis.

Treatment

Primary treatment depends on correction of the cause.

Supportive treatment is by the application of a keratolytic agent (e.g. salicylic acid ointment).

4- Parakeratosis

Parakeratosis, a skin condition characterized by incomplete keratinization of epithelial cells. It is a nutritional deficiency disease of 6- to 16-week-old pigs characterized by lesions of the superficial layers of the epidermis. It is a metabolic disturbance resulting from a deficiency of zinc or inadequate absorption of zinc due to an excess of calcium, phytates (phytic acid), or other chelating agents in the diet. Predisposing factors include rapid growth, deficiency of essential fatty acids, or malabsorption due to GI diseases.

Caused by:

- Nonspecific chronic.
- Inflammation of cellular epidermis.
- Associated with dietary deficiency.

Pathogenesis

The initial lesion comprises edema of the prickle cell layer, dilatation of the inter cellular lymphatic's, 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.

Clinical signs

The lesions may be **extensive** and **diffuse** but are often confined to the flexor aspects of joints (referred to historically in horses as mallenders and sallenders) . 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, and their removal leaves a raw, red surface. Hyperkeratosis scales are thin, dry and accompany an intact, normal skin.

Diagnosis

Confirmation of a diagnosis of parakeratosis is by the identification of imperfect keratinization in a histopathological examination of a biopsy or a skin section at necropsy.

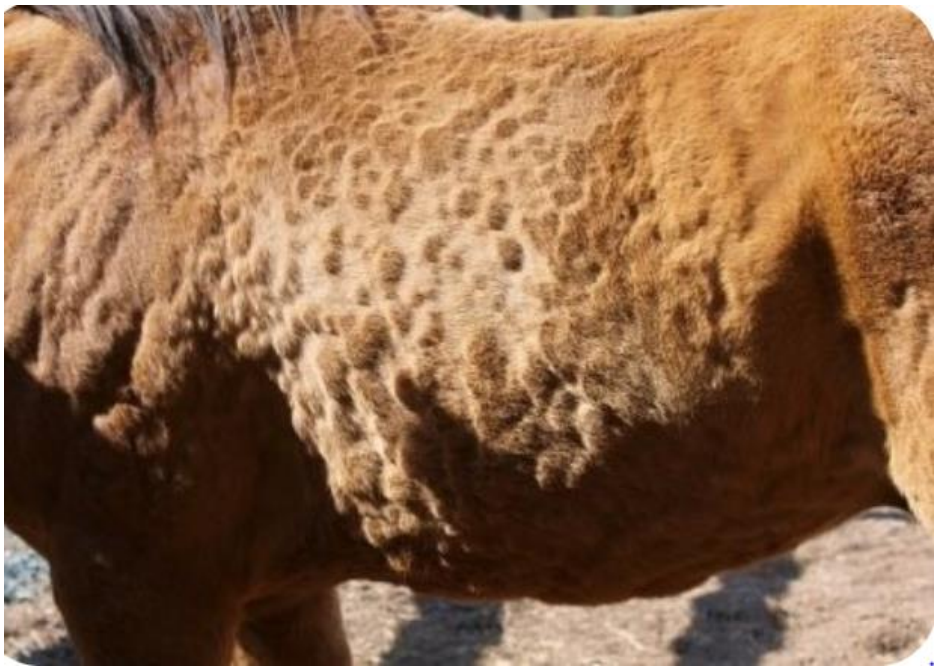
Differential diagnosis

1- Hyperkeratosis. 2- Pachyderm. 3- Ringworm. 4-Inherited ichthyosis. 5- Inherited edema disease in cattle. 6-Inherited epidermal dysplasia.

Treatment

Primary treatment requires correction of any nutritional deficiency. **Supportive treatment** includes 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.

5- URTICARIA



An allergic condition characterized by cutaneous wheals. It is most common in horses.

Etiology

1- Primary urticaria : Results directly from the effect of the pathogen, e.g.:

- Insect stings
- Contact with stinging plants
- Ingestion of unusual food, with the allergen, usually a protein.
- Occasionally an unusual feed item, e.g. garlic to a horse After a recent change of diet .
- Administration of a particular drug, e.g. penicillin; possibly guaifenesin or other anesthetic agent .
- Allergic reaction in cattle 8 days following vaccination for foot-and mouth diseases.
- Death of warble fly larvae in tissue .
- Milk allergy when Jersey cows are dried off Transfusion reaction.

2- Secondary urticaria

occurs as part of a syndrome, e.g.: Respiratory tract infections in horses, including strangles and the upper respiratory tract viral infections.

Pathogenesis:-

An allergic reaction. ⟶ degranulation of mast cells ⟶ liberation of chemical mediators inflammation, resulting in the subsequent development of dermal edema. A primary dilatation of capillaries causes cutaneous erythema.

Exudation from the damaged capillary walls causes local edema in the dermis and a wheal develops. Dermis, and sometimes the epidermis, is involved.

(Note) :In extreme cases the wheals may expand to become seromass, when they may ulcerate and discharge. The lesions of urticaria usually resolve in 12-24 hours but in recurrent urticaria an affected horse may have persistent and chronic eruption of lesions over a period of days or months.

Clinical feature

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 0.5-5 cm in diameter, with a flat top, and are tense to the touch.
3) There is often no itching, except with plant or insect stings, nor discontinuity of the epithelial surface, exudation or weeping.
4) Pallor of the skin in wheals can be observed only in un pigmented skin.
5) The affected areas become hairless and the wheals exude serum and become scabbed over.
6) Edema of the legs is common and vesicles occur on the teats. The lesions appear 8-12 weeks postvaccination and may persist for 3-5 weeks.
7) Loss of body weight and lymphadenopathy.

Clinical pathology

a. Intradermal skin tests to detect the presence of hypersensitivity are of little value because many normal horses, as well as those with urticaria,

will respond positively to injected or topically applied allergens. Also, reactions usually occur within the first 24 hours after the injection, but the interval is very erratic.

b- Intrademal tests in horses without atopy and horses with atopic dennatitis or recurrent urticaria using environmental allergens indicate a greater number of positive reactions for intradennal tests in horses with atopic dennatitis or recurrent urticaria, compared with horses without atopy.

c- Biopsies show that tissue histamine levels are increased and there is a local accumulation of eosinophils.

d- Blood histamine levels and eosinophil counts may also show transient elevation.

Treatment

- **Primary treatment** A change of diet and environment, especially exposure to the causal insects or plants, is standard practice. Spontaneous recovery is common.
- **Supportive treatment** Corticosteroids, antihistamines, or epinephrine by parenteral injection provide the best and most rational treahnent, especially in the relief of the pruritus, which can be atmoying in some cases.
- **The local application** of cooling astringent lotions such as calamine or white lotion or a dilute solution of sodium bicarbonate is favored.
- In large animal practice parenteral injections of calcium salts are used with apparently good results. Long-term medical management of persistent urticaria involves the administration of corticosteroids and or antihistamines.

- Oral administration or prednisone or prednisolone at the lowest possible dose on alternate days is the method of choice. The antihistamine of choice is oral hydroxyzine hydrochloride initially at 600 mg three times daily, followed by gradual reduction to a minimum maintenance dose required to keep the horse free of lesions.

6- **Impetigo**

Superficial eruption of thin -walled, small vesicles, surrounded by a zone of erythema, that develop into pustules, then rupture to form scabs.

Causes

A staphylococcus: The only specific examples of impetigo in large animals are:

- Udder impetigo of cows.
- Infectious dermatitis or' contagious pyoderma' of baby pigs associated with unspecified streptococci and staphylococci.

Pathogenesis

The causative organism and 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.

Clinical feature

- Small (3-6 mm) vesicles appear chiefly on the relatively hairless parts of the body and do not become confluent.
- In the early stages each vesicle is surrounded by a narrow zone of erythema.
- No irritation is evident.
- Vesicle rupture occurs readily but some persist as yellow scabs.
- Involvement of hair follicles is common and leads to the development of acne and deeper, more extensive lesions.
- Individual lesions heal rapidly in about a week but successive crops of vesicles may prolong the duration of the disease.

Diagnosis

Confirmation of the diagnosis is by culture of vesicular fluid and identification of the causative bacterium and its sensitivity.

Differential diagnosis

- Cowpox, (the lesions occur almost exclusively on the teats and pass through the characteristic stages of pox).
- Pseudocowpox.

Treatment

Primary treatment

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.

7- Pachyderma

Pachyderma including scleroderma, is thickening of the skin affecting all layers, often including subcutaneous tissue, and usually localized but often extensive as in lymphangitis and greasy heel in horses.

Causes :-There are no specific causes, most cases being due to nonspecific chronic or recurrent inflammation.

Pathogenesis :- In affected areas the hair coat is thin or absent and the skin is thicker and tougher than usual. It appears tight and, because of its thickness and reduced volume of subcutaneous tissue, cannot be picked into folds or moved easily over underlying tissue.

The main signs:-

- The skin surface is unbroken
- No lesions
- No crusts or scabs as in parakeratosis and hyperkeratosis.

Diagnosis

Confirmation of the diagnosis depends cells in all layers are usually on histopathological examination of a biopsy. The normal but the individual layers are increased in thickness. There is hypertrophy of the prickle cell layer of the epidermis and enlargement of the interpapillary processes.

Differential diagnosis

1- Parakeratosis 2- Cutaneous neoplasia 3- Papillomatosis

Treatment

Primary treatment requires **removal** of the causal irritation but in well-established cases little improvement can be anticipated, and **surgical** removal may be a practical solution when the area is small. In early cases **local or systemic** corticosteroids may effect a recovery.