Diseases of Skin
And its associated Structures

Dermatology is the science, which deals with the study of the skin or coat of the animal either from the normal physiological and anatomical picture, or from the diseases, which affect the skin and its effects on the animal health and production. The skin forms the largest single organ of the body, performing not only a supportive anatomical role but also a wide variety of important physiological functions essential to the well being of the animal.

The skin is a heterogeneous organ, which serves as principal medium of communication between the animal and environment. The anatomical, histological and physiological features of the skin vary between species & breeds.

Anatomical and Histological Consideration
The skin is divided into two layers an outer layer which is called epidermis and an inner layer known as dermis, and associated structures which known as skin appendages including hairs, horns, claws, nails, hooves. Sebaceous and sweet glands all develop from the epidermis in addition to the smooth muscle fibers attached to hair follicles and distributed in the dermis.

In general epidermis of mammals is composed of 5 layers as the following:-
1. Stratum germinativum or basal layer.
2. Stratum spinosum or stratum Malpighii.
3. Stratum granulosum.
4. Stratum lucidum.
5. Stratum corneum.

While the dermis composed of bundles of collagen, elastic and reticular fibers within a homogenous ground of sulfuric and hyaluronic acids within the matrix is population of Fibroblast, most cells and histocytes, blood vessels, nerve endings are present.

Physiological Consideration
The main functions of the skin are:
1. Physical protection from trauma, temperature variations, invasion of microorganisms and over exposure to sunlight.
2. The skin acts as biological barrier, which prevents the passage of harmful agents into the body. Mechanical protection of the keratinized structures against the environment.
3. Skin maintains the internal conditions of the individual so it acts as a barrier for water and electrolytes and prevents their loss.
4. Synthesizing vitamin D by the action of ultra violet rays and transforming steroids to vitamin D.
5. Aids in maintaining the normal Blood proteins by the action of peripheral vascular dynamics.
6. Help in recognition of foreign protein as contact allergens and venom and stimulate the antibody production. this is due to the presence of specific immunoglobulin movement into and through the epidermis.

**Skin lesions and their terminology**

**Nodules**
Nodule is a circumscribed, solid elevation greater then 1 cm in diameter that does not deform when palpated.
Nodule extends into the deeper layers of the skin, and it results from cellular infiltrates into the dermis and subcutis.

**Ulcers**
An ulcer is a cutaneous defect resulting from a complete loss of the epidermis and usually part of the underlying tissues.

**Erosion**
Erosion is a cutaneous defect resulting from partial loss of the epidermis that does not penetrate beneath the basal laminar zone.

**Papules**
A papule is a solid, circumscribed, elevated lesion up to 1 cm in diameter.
Papules are essentially small nodules that do not extend beneath the dermis.

**Pustules**
A pustule is a pus-fined, fluctuant, circumscribed, and elevated accumulation of pus up to 1 cm in diameter.

**Vesicles**
A Vesicle is a fluid-filled, a cellular, circumscribed, elevated lesion up to 1 cm in diameter. While, a bulla is a vesicle that is greater than 1 cm in diameter.

**Scaling**
Scale is a visible accumulation of fragments of the horny layer of the skin (Stratum corneum). It represents the final product of epidermal keratinization.
Histologically, scale is recognized as hyperkeratosis, which may be either parakeratosis or orthokeratosis. Grossly, it varies in appearance (color), consistency, and adherence.

**Crusts**
Crusts are dried exudate that adheres to the skin surface and hair. Crusts often cover erosions or ulcer; crusts are composed of serum, cells, fibrin, and infectious agents. Dirt and medications.

**Diseases of the skin and its associated structures**

**Importance of skin diseases**
- As well as being involved directly in a variety of disease process, the skin and coat are influenced indirectly by the general health status of the individual animal.
• The incidence of skin diseases in domestic animals is high and it is important to remember that some skin diseases are contagious, so that prompt recognition is important and essential in order to prevent further dissemination of the infection, and to assist control.
• The risk to persons handling the animals affected with certain parasitic disease of skin is an important public health responsibility for the veterinary clinician as mange and ringworm.
• Skin diseases cause restlessness at least to the animal and decrease body weight gain and decreased its production.

Classification of skin diseases
(1) According of its origin:
   1- Primary skin diseases:
   In this type of diseases initially at least the lesions are restricted to skin and its associated structures, spread to other tissues may occurs later as secondary complications. It is evidenced by the clinical examination, which reveals that the lesions are restricted to skin without systemic reactions.
   2- Secondary skin diseases:
   In this type the lesions occur as the result of extension of the disease process from another organ or tissues other than the skin, system reactions are present with cutaneous lesions.

(2) According to the causative agent:
   (A) Non-infectious skin diseases includes:
   **Diseases affect Epidermis**
   1- Pityriasis.
   2- Parakeratosis.
   3- Hyperkeratosis.
   4- Pachydermia.
   5- Impetigo.
   6- Urticaria.
   7- Dermatitis.
   8- Eczema.
   9- Photosensitization.
   **Diseases affect skin appendages**
   1- Alopecia.
   2- Achromotrichia.
   3- Seborrhea.
   4- Acne.

Disease of subcutis:
1. Subcutaneous edema.
2. Angio neurotic edema.
3. Subcutaneous emphysema.
4. Lymphangitis.
5. Skin-Hemorrhages.
7. Skin. Abscess.

(B) Infectious skin diseases includes:
   (A) Viral diseases:
   1- Cowpox - pseudo cow pox.
   2- Contagious pustular dermatitis.
   3- Swinepox - sheep, goat pox.
   4- Bovine ulcerative mammilitis.
   5- Warts = viral papillomatosis.
   6- Epuine sarcoides.
   7- Viral popular dermatitis.
   8- Coital exanthema.
(B) Bacterial diseases:
1- Dermatophilosis = mud fever. 2- Impetigo.
3- Contagious acne. 4- Streptothrichosis.
5- Ulcerative Lymphangitis. 6- Glanders “Farcy”.
7- Subcutaneous abscesses.

(C) Mystic diseases:
1- Ring worm.
2- Epizootic Lymphangitis.
3- Sporothricosis.

(D) Parasitic diseases:
1- Lice infestation.
2- Ticks infestation.
3- Mange.
   - Psoropite mange (Body mange, ear mange).
   - Sarcoptic mange (Red mange, Barn itch).
   - Demodectic mange (Follicular mange).
   - Chorioptic mange (Leg mange, tail mange).
4- Other diseases.
   - Parafilaria multipilosa.
   - Cutaneous neoplasms.
   - Granulomatous lesions.
   - Congenital skin lesions.

DIAGNOSTIC METHODS IN DERMATOLOGY:
The sequence of procedures in laying the foundations for an accurate diagnosis is:
1- Case history,
2- Physical examination,
3- Skin scrapings.
4- Skin biopsy.
5- Initial diagnostic tests.
6- Differential diagnosis

(I) History:
A detailed history is obtained from the owner or person having most contact with the animal. A good case history provides assistance in diagnosis and treatment. The history must not be limited to cutaneous symptoms but should include information on other systems.

General history taking:
(1) Age: Some skin diseases are associated with specific age group as for example lymphadenitis.
(2) Sex: In females, skin disorders resulting from estrogenic imbalance frequently develop. In males testicular tumors cause specific skin lesions as hypotrichosis “Hairlessness” due to sertoli-cell tumor, which is one type of Alopecia.
(3) Species and breed: Some species of animals and even certain breeds develop skin lesions specifically.
(4) Environment: including climate and geographical conditions which effect on the skin, such as distribution of trace elements in the soil may lead to many of skin lesions. Also allergic skin disorder as in photosensitization may be relate to environmental condition.

(5) Nutritional conditions: Some skin lesions are developed from deficient nutrient in some vitamins, minerals and tract elements as facial eczema due to vit. A and zinc deficiency and may due to excessive feeding Alopecia in a yearling bullock suffering from advanced molybdenosis.

**Principals of History taking “In Relation to skin only”:-**

1- Has the patient previously suffered a disease? If so, what is the diagnosis?
2- Do of other animals of family have a similar skin disease?
3- Have the lesions been localized or generalized in many areas?
4- What has the duration of the lesions?
5- What is the usual behavior of the patient?
6- What type of the patient nutrient and source of water?
7- What is the patient history concerning external and internal parasite?

(2) **Physical Examination:**

A complete physical examination is carried out and the status of all body system evaluated and nored. All parts of the skin are examined; the coat being parted and skin palpated where necessary. Good lighting and use of the magnifying lens are essential. The condition of the coat and the nature and distribution of any lesions are noted and it is important to recognize the different types of skin lesions and to describe them accurately.

(3) **Differential diagnosis and selection of diagnostic tests:**

Careful consideration of the history, together with the results of the physical examination, will suggest a number of possible causes of condition. The diagnostic procedures must be selected, which will confirm or eliminate these possibilities. It is important to differentiate primary and secondary skin disease.

* **Diagnostic tests include:**

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**Wood’s lamp illumination:**-

The wood’s lamp illumination depends on a source of ultra-violet radiation at wavelength, which excites a characteristic apple-green fluorescence in about 50% of naturally occurring Microsporum canis infection, and other Microsporum species also fluoresce. It is seen only in infection of actively – growing hair. The disadvantages of
this method are, it may give + ve result with other chemical agents as tetracycline, and failure to demonstrate such fluorescence dose not rule out dermatophytes.

**Skin Scrapings:**
The skin scraping is one of the most valuable and commonly used tests in veterinary dermatology, confirming the diagnosis of the ectoparasites and dermatophytes. The hair, superficial scales, epidermis and contents of the hair follicle mouths may be sampled by this technique.

**Protocol of skin scrapings:**
1. Select the area of scraping with great care to the predilection site for the disease either ectoparasites or dermatophytes.
2. The hair should be firstly trimmed short.
3. Skin should be gently wiped with swab moistened with sterile water.
4. Scraping is done with scalp blade held firmly between the thumb and first two fingers of one hand at angle of about 50% to the skin and drawn firmly across the surface towards the operator. The surrounding skin is tensed with fingers of other hand.
5. Scraping is continued until the first signs of bleeding appear. Moistening the skin with water, mineral oil or glycerin for adherence of the scrapings to the blade.
6. Microscopical examination of the scrapings.
   - Scrapings are suspended in a drop of oil on microscope slide, and covered with cover slip, and examined under the x 10 objective. Microscope condenser should be lowered to increase the contrast.
   - Scrapings are collected dry or moistened with water and suspended on slide in 20% potassium hydroxide.
   - Suspension is warmed to accelerate clearing.
   - Cover slip is applied and the preparation is examined under the x 40 objectives.
   - Scrapings are collected in test tube or small beaker and add 4-10% sodium or potassium hydroxide.
   - Heat gently, but not boil, until the hair is dissolved for about 5 minutes. “If the sample is boiled the parasite will be transparent and difficult to diagnosis”.
   - Maceration overnight without heat may be sufficient.
   - Allow the tube to stand for minutes and cooling.
   - Centrifuge the sample and examine the sediment.
   - Sample from the bottom by glass rode or dropper and transferred to slide than cover with cover glass and examined microscopically under the low power.
   - Scrapings can be collected dry in sealed paper envelope and then cultured directly for isolation of dermatophytes on specific media.

**Hair plucking:**
Hairs from the chosen site are grasped firmly with forceps and plucked and it may be inoculated directly onto specific media for mycological isolation and identification. Or examined under the microscope with low power objective x 10 and the hairs are mounted between glass slides held together with tape. Or under x 40 objective and hair held with mineral oil gives better resolution.
Coat brushing:
It is useful where the skin lesions are diffuse and infection of the hair or superficial stratum corneum is suspected. Coat brushings enable the loose hairs, scruff and crusts from large areas of the skin to be collected.

- The animal is placed on sheet of clean paper.
- The coat ruffled with coarse brush causing any loose material to be fall onto the paper.
- The collected materials are examined under the microscope under x 10 objectives in between glass slides.
- Some debris is screened with wood’s lamp.
- Sample may be put on slide and examined microscopically in mineral oil or in 20% potassium hydroxide.
- If fungal or dermatophytes are suspected the brush cultures on specific media.

Swab and Crust samples:
Cotton swabs are commonly used to sample pustular or exudative lesions for smear preparation and isolation of bacteria and fungi e.g. Candida from the skin.

- The hair is first clipped from around the lesions.
- Gentle cleaning with 70% Alcohol.
- Pustules are opened with the tip of sterile needle.
- The adjacent skin is gently squeezed.
- The emerging pus collected on the tip of the swab and avoiding contact with the skin.
- The furuncular and scabby lesions are also sampled as pustules but scabs are a potent source of microorganism as poxvirus and dermatophilosis. Portion of scabs may be emuls.

Smears and wet preparations:
Smears and wet preparations provide a rapid and relatively simple means for the demonstration of m.o. and host cells in skin lesions. It is particularly useful in demonstration of yeast and neoplastic cells.

Smears may be prepared in three ways:-
1. Direct impression smears are made by pressing the surface of moist skin lesions, the base of freshly removed scab or the cut surface of a biopsy specimen causing cells and exudate to adhere to it.
2. Smear also can be made from pus or exudate taken from the lesion and spread thinly onto the slide.
3. Samples are collected on swab by scraping the affected skin. or by aspiration using needle and syringe.
4. They are smeared onto the slide with swab or using bacteriological loop.

Smears may also made from emulsified scabs:
- Slides should be cleaned with alcohol prior to use to promote adherence and even distribution of the material.
• The smears are air-dried, fixed by flooding with alcohol for one minute and allowed to dry.
• Staining smear with an appropriate technique depending on the features, which are to be demonstrated. Most common stains are: Gram’s, methyleneblue, Giemsa.
• Wet preparations are unstained specimens prepares from exudate or scab emulsions; as described by many researchers.
• Drop of specimen is placed on the slide.
• Normal saline is added to dilute the material or as mounting medium.
• Cover slip is applied.
• Examination under the microscope by phase-contrast illumination or with the condenser lowered.

Biopsy samples:
These samples are usually obtained for histopathology:
• Local anesthesia.
• Hair is clipped with scissors.
• Cleaning with 70% alcohol.
• Incision of the skin or punch method is applied.
• Specimen put in 10% formaline for at least 24 hours.

Blood samples:
• Changes in the cellular or biochemical composition of the blood are useful in confirming or ruling out differential diagnosis in dermatology.
• Specialized tests as hormonal assays, and serological tests may be used to identify specific conditions.
• Collection of blood.

(A) Clotted blood or serum:
• Cleaning the site of vein puncture.
• Obtaining the blood in the containers as centrifuge tubes vials, …
• The container is left to clotted in sunlight in sleep manner to give large surface area for oozing of serum.
• Then centrifuge the sample for obtaining maximum amount of serum.
• Transfer the serum into other tubes or vials and closed then preserved in deep freezer.
• Serum is collected for demonstration of biochemical composition such as minerals including: Copper, Zinc, Manganese, Sulfur and other elements…. Iron and calcium.

(B) Whole Blood sample:
Blood samples are obtained by vein puncture using sterile needle and adding anticoagulants as heparin and EDTA. Whole blood samples are obtained for detection the cellular changes, which may be the cause, are as result of diseased skin. This changes to be detected necessitate the following:-
• Erythrocytic count.
• Total leucocytic count.
• Differential leucocytic count.
SPECIAL PATHOLOGY:-

The reaction of the skin to noxious stimuli varies with the severity and depth of injury. In the corium or dermis the reaction is the same as that of other tissues due to presence of blood vessels, nerve fibers, lymphatic vessels and connective tissues. The epidermis due to purely cellular composition reacts differently.

**(A) Acute Reaction:**

If the reaction is acute, the development of lesions begins with swelling and edema of prickle cell layer and so called spongiosis. If the edema is severe enough, cell rupture and fluid collects as foci which gradually emerge through the stratum corneum and appear as vesicles. Should the foci rupture before reaching surface, the result is weeping of the area.

**(B) Sub-Acute Reaction:**

The intercellular edema interferes with the normal functions of the granular cells in the prickle layer and gives rise to abnormal formation of cornified epithelium, and result in thickening of epidermis. All layer are affected specially stratum corneum because of improper keratinization and failure of exfoliation this is so called parakeratosis; it may be accompanied by pronounced thickening of prickle cell layer with prolongation of interpapillary processes and so called acanthosis, the disease state in this case named pachydermia.

Acanthosis in association with the deposition of keratin pigment described as Acanthosis nigricans, which is common in dog and human, associated with thyroid dysfunction.

**Skin diseases due to allergens:**

When an allergen is applied on sensitized skin, local rise in histamine levels leading to an accumulation of eosinophils. If the histamine level increased about the detoxifying capacity of eosinophils, it will escape to vascular system and blood level of histamine rise. This is transitory and be overcome in about 1-2 hours after removal of allergen.

- Examination of histamine level or eosinophils count may be of diagnostic value.
- The local skin reaction to the allergen is due to the vascularity effect of histamine.
- If the reaction is severe enough other organs may showing histamine toxicity, this also may occurs when the allergens ingested which produce reactions on other end organs and including skin.

**PRINCIPLES OF TREATMENT OF DISEASES OF THE SKIN**

1. Removal of hair coat and debris to enable topical applications to come into contact with the causative agent is preferable.
2. Accurate diagnosis must be preceding the selection of drugs.
3. In bacterial diseases sensitivity tests on culture is advisable.
4. In allergic diseases and photosensitization may be impossible and the only symptomatic treatment is the solution.
5. Removal of causative agents by specific treatment for each once.
6. Prevent secondary infection by using bacteriostatic drugs.
7. Prevent further damage from scratching by using local anesthetic ointments or centrally acting sedatives.
8. When large area of skin is involved, prevent absorption of toxic subs. by continuous irrigation or application of absorptive dressing.
9. In cases of fluid losses; it must be given as isotonic fluid by the parenteral administration.
10. Good ration specially protein and sulfur containing amino acids to help in repair of skin.

**SKIN DISEASES**

(1) **PITYRIASIS = DANDRUFF:**
It is non-infectious condition characterized by the presence of bran-like scales on the skin surface.

**Etiology:**
1. Hypovitaminosis A and B especially riboflavin and nicotinic acid mainly in pigs.
2. Nutritional deficiency acids as linolenic acid.
3. Poisoning by iodine which causing fatty acid deficiency.
4. Lice, flea and mange infestations.
5. May be with ringworm.

**Pathogenesis:**
The scales are keratinized epithelial cells and these are sometimes softened and become greasy due to exudation of sebum or serum. Avitaminosis A results in overproduction of keratinized epidermis. Excessive desquamation due to parasitic infestation is another way of pathogenesis and developing scales.

**Diagnosis:**
Primary Pityriasis depends upon the examination of skin scrapings. Differentiation from hyper-and parakeratosis. Skin scrapings to eliminate parasites and Fungi.

**Treatment:**
1. Correction of primary agents.
2. Using of balanced ration emollient ointment and alcoholic lotion.
3. Salicylic acid incorporated in ointment and lotions.

(2) **HYPERKEATOSIS**
It is the accumulation of excessive keratinized epithelial cells on the surface of the skin Resulting in thickening of the skin with or without hair loss.

**Etiology**
1- Localized at pressure points as elbow when the animal lays habitually on hard surface “Mechanical”.
2- Chronic poisoning with arsenic compounds.
3- Poisoning with highly chlorinated naphthalene compound used in industry as in wool preservation.
4- Inherited as congenital ichthyosis = fish – scale disease of cattle. ( Alopecia with plates of horny layer allover the skin; this occurs in newly born animals specially calves).

**Pathogenesis**
- Local compression leads to accumulation of keratinized epithelial cells.
- Excessive keratinization of epithelial cells and intercellular bridges and hypertrophy of stratum corneum.
- In cases of poisoning with chlorinated naphthalene, it causes deficiency of the granular layer of epidermis and causing atrophy of epithelial cells.

**Clinical finding**
1- The skin becomes thicker than normal and usually hairless and corrugated.
2- Skin becomes dry and fissures develop in grid-like fashion.
3- Secondary infection through the fissures when the skin is wet.

**Diagnosis**
- Differentiation from parakeratosis.
- Skin scrapings to eliminate ectoparasites and fungal infection.
- Histological examination of skin biopsy to detect the thickened stratum corneum.

**Treatment**
1- Correction of the primary cause.
2- Use of balanced ration, emollient ointment and alcoholic lotion.
3- Salicylic acid incorporated in ointment and lotions.
4- Using of vitamin A as 6000 I.U

(3) **PARAKERATOSIS**
It is a condition of the skin in which keratinization of epithelial cells is incomplete.

**Etiology**
1- Non-specific chronic inflammation of cellular epithelium which leads to faulty keratinization of horny cells.
2- Dietary deficiency of saturated fatty acids. Additional dietary zinc deficiency along with copper alleviates the condition, which is effectively prevented by supplements of soybean oil or some other suitable source of linoleic acid.
3- Inherited dermatosis vegetans in pigs.

**Pathogenesis**
Edema of prickle cell layer, with dilatation of intercellular lymphatic vessels and leucocytic infiltration lead to Imperfect keratinization at granular epithelium. Layer of epidermis produces sticky and soft horn cells, which retain their nuclei and they tend to stick from large masses and either still fixed to underlying tissues or may fall off as forge scales.

**Clinical Findings**
1- Lesions may be diffuse and extensive but often confined at flexor aspects of joints.
2- Reddening, thickening and gray coloration of skin.
3- Cracks and fissures also develop and removal of scales leave raw and red surface.

**Diagnosis**
1- Histologically: imperfect keratinization is evident.
2- When the scales are removed it leaves raw and red area so differentiation from hyperkeratosis will be easy.

**Treatment**
1- The deficiency in ration must be corrected by addition of zinc continuously using zinc sulphate or zinc oxide.
2- Removal of scales by using keratolytic ointment as that of salicylic acid ointment or by warm soapy water.
3- Local application of astringents as white lotion paste.

(4) URTICARIA (NETTLE RASH)

Urticaria is an allergic condition characterized by the appearance of wheals on the skin surface.

**Etiology**

It may be a primary lesion resulting from the direct effect of pathogen on the skin, or may be secondary as part of a syndrome.

(A) Primary Urticaria due to:

1- Insect bites.
2- Ingestion of unusual food mainly protein and sudden changes of diet is a predisposing factor.
3- Drug administration (e.g. penicillin).
4- Death of warble fly larvae in tissues.

(B) Secondary Urticaria due to:

1- Alimentary tract disturbances in horse.
2- Respiratory tract infection in horse.
3- Along the course of some disease.

**Pathogenesis**

Urticaria represents a type of immediate hypersensitivity on which the binding of antibody with antigen results in release of Histamine.

- Primary dilatation of capillaries causes erythema of skin.
- Exudation from damaged capillaries leads to local edema of epidermis with swelling and pallor due to compression of capillaries.
- The lesions remain red at the edges and only the epidermis is involved.

**Clinical Findings**

1- Lesion develop rapidly, large in numbers, ranges from 0.5 - 5 cm diameter with flat-topped steep-sided plaques, and tense to touch.
2- No exudation or weeping occurs; No itching except with insect bite.
3- Plaques mainly found at back, flank, neck and legs.
4- It may subside in 24 hours or last up to 5 days.

**Clinical Pathology**

There is an increase of histamine level and local increase of eosinophils count.

**Diagnosis**

Urticaria must be differentiated from angioneurotic edema; In cases of edema, the subcutaneous tissues are involved.

**Treatment**

1- Antihistaminic with parenteral administration of adrenaline.
2- A mild purgative and corticosteroids may be used.
3- Local astringent as white lotion, calamine lotion or dilute solution of sodium bicarbonate.
4- In large animals, parenteral administration of calcium salts.
Changing the diet is helpful in cases of food allergy.

(5) ECZEMA: ECZEMATOUS DERMATOSES:
It is an inflammatory reaction of the epidermal cell to exogenous or endogenous substances to which cells are sensitized. This occurs when the skin is in contact with allergens either applied on skin and so called exogenous or arise from blood stream and known as endogenous allergen.

Etiology

(A) Predisposing Factors
1- Nutritional deficiency, trauma and chemicals.
2- Genetic causes.
3- Prolonged soiling, dampness and accumulation of debris.
4- Constant scratching due to external parasites.

(B) Exogenous Allergens:
➢ Using of chemicals as antiseptics and disinfectants.
➢ Some Ectoparasites as flea-saliva in dog and cat, which cause hypersensitivity of skin.

(B) Endogenous Allergens:
1- These are substances, which are ingested and absorbed through the gut and introduced to blood stream and affect on the skin indirectly ingestion proteins.

Pathogenesis
Primary lesion is erythema, Spongiosis due to in intra and intercellular edema forming vesicles which is characteristic for eczema, this leads to rupture of vesicles and causes weeping of skin with formation of scabs. This occurs in acute stage of eczema and may disappear rapidly Chronic form may persist accompanied with parakeratosis and pachydermia.

Clinical findings
True eczema is rare in large animals.

In typical form
1- Erythema, papules and vesicles on the back of animal, weeping of the surface mainly in longhaired dogs.
2- Itching, scratching and rubbing which aggravate the condition.

In chronic form
1- Alopecia in many areas of skin.
2- Scaling and hypertrophy of skin.
3- Pachydermia but no discontinuity of skin.

Diagnosis
The clinician must define the cause and exclude other disease but it is difficult.

Treatment
1- Improve environmental condition of animal, change of bedding and diet, will be useful.
2- Treatment of internal and external parasites.
3- Sedatives in early stags.
4- Antihistaminic.
5- Corticosteroids as anti-inflammatory.
6- Local application of astringent on weeping area, In addition to local anesthetic agent.

(6) ALOPECIA
Alopecia is deficiency of hair or wool coat. It is a manifestation of many disease as eczema, dermatitis, and mange.

Etiology
In most of species, hair loss occurs normally in the spring and autumn for the coat to be changed. However, hair loss may be due to failure of follicle to produce a fiber or it may due to damage hairs previously.

(A) Failure of follicle to produce hair fibers
1- Inherited hypotrichosis, symmetrical Alopecia. The skin in the affected area becomes completely bald.
2- Congenital hypothyroidism “goiter” due to deficiency of iodine in dam.
3- Congenital, after viral infection of dam as in Bovine Viral Diarrhea in cattle and sheep.
4- Infection of follicles or neurogenic due to damage of peripheral nerve.
5- Sertoli cell tumors in Male dog.

(B) Damage of produced fibers
It is mainly symptomatic Alopecia due to other diseases.
1- Dermatomycosis as ringworm, (dermatophytosis).
2- Poisoning of thallium or molybdenum.
3- Deficiency of copper and zinc and vit. A, vit. E.
4- Excessive palm - whale or soybean in milk replaces of calves usually result in weakness of hair fibers.
5- Traumatic as in sweat itch of horses.

In case of symptomatic alopecia it is a temporary case or condition that will be improved by removal of the original causes.

Pathogenesis
- In many of metabolic alopecia there is weakness of fibers, which degenerated rapidly.
- In congenital Alopecia, there is failure in follicles to form fibers.
- Damages of nerve ending and blood capillaries are impaired.
- Chemical depilation may occur by cytotoxic agents – cytoplasmic degeneration in follicle of hair (chemotherapy).

Clinical findings
- Stumps of breakage or new-formed fibers are seen.
- Skin may be shiny and thinner than normal (this is true only when fibers fail to grow).
- There are manifestations of primary disease.
- Scratching or rubbing.

Diagnosis
Clinical signs.

Treatment
1- Treatment of primary causes.
2- Improve the diet with balanced quantities.
3- Improvement of blood supply of skin.
4- In gonadal disorders, castration or administration of gonadal hormones might be useful in reactivation.

(7) Dermatitis
The term dermatitis include those condition characterized by inflammation of the deeper layer of the skin, including the lymphatic with secondary involvement of epidermis.

Etiology
The causes and types of dermatitis in all animals classified into:
1- **Bacterial dermatitis:**
- Due to invasion of bacteria as in udder impetigo in cattle.
- Pyoderma due to Staph. aureus.

2- **Mycotic dermatitis**
- Dermatophilus congolensis in sheep, cattle, and horses.
- Ringworm
- Strawberry foot Rot, (dermatophilus pedis).

3- **Viral dermatitis**
- Poxvirus infection.
- Contagious pustular dermatitis.
- Lumpy skin disease in cattle.
- Foot & Mouth disease, vesicular exanthema.
- Vesicular stomatitis – mucosal disease.
- Blue tongue – bovine malignant catarrh.

4- **Parasitic dermatitis**
- Mange and other mites.
- Myiasis of hypoderma and others.

5- **Nutritional dermatitis**
- Deficiency of vit. B. complex, vitamin A, zinc, nicotinic acid, riboflavin, biotin, pantotenice acid.

6- **Physical dermatitis**
- Sunburn and photosensitization.

7- **Chemical dermatitis**
- Arsenical compound.
- Potassium – mercuric iodide.

8- **Allergic dermatitis**

**Pathogenesis**
- Involvement of deeper layers including blood vessels, lymphatic and epidermis.
- Black necrosis at the site of inflammation.
- Erythema with other factors causes an increase in the thickness of skin accompanied by edema.
- Pain or itching.

**Clinical findings**
1- Erythema and hotness of the affected part of the skin.
2- Vesicular lesion and edema of skin.
3- Scab formation, necrosis or gangrene may occur.
4- Phlegmon may be developed, distinctive suppurative lesions.
5- Tenderness.
6- Systemic reactions may occur (such as toxemia, septicemia).
7- Secondary bacterial infection may result in pus formation.

**Treatment**
Treatment of the primary cause.
Supportive treatment by local and systemic application.
Antihistaminic.
Astringents.
Anesthetic agents.
Antibiotic administration to prevent secondary complication.
Parenteral fluid should be administered in case of shock.
High protein diet.

(8) PHOTOSENSITIZATION (LIGHT SENSITIZATION = SUNBURN)
It is secondary damage following injury to superficial layer of relatively unpigmented skin by the energy released from interaction between light of certain wavelength and photodynamic agents. Dermatitis develops when the sensitized skin is exposed to strong light.

Etiology and types of photosensitization
If the photodynamic agents are present in sufficient concentration in the skin, dermatitis occurs when skin is exposed to light.

(1) Primary photosentization
This type is due to ingestion of exogenous photodynamic agents usually in lush green plants and the animals affected within 5 days from going onto pasture and symptoms are disappeared when the animal is removed away from pasture and this differs according to the susceptibility of animals. The most active form occurs naturally in plants and causes photosensitization are:
- Miscellaneous substance as acridin dyes and rose Bengal.
- Fagopyrin in seeds of dried plants of buck wheat.
- Perloline from perennial rye grass.

(2) Photosensitization due to aberrant pigment synthesis:
May be due to production of abnormal metabolism, the only example is inherited congenital porphyries in domestic animals due to excessive production of prophyrins substance, Which is the photodynamic agent.

(3) Hepatogenous photosensitization
This is due to accumulation of normal metabolic products as a result of failure to excretion through the liver.

- Normally a photosensitizing substance, which is known, as phylloerythrin is normal end product of chlorophyll metabolism and excreted in the bile.
- When the level of phylloerythrin is increased due to hepatitis or cholangio hepatitis and other cause of obstruction of biliary passage and reach in level in skin to the point, which make it sensitive to light.
- This is the most common form on all animals especially that fed on green pasture also occurs in other animals fed only on hay and other stored feed.
- Fungus on perennial ryegrass.
- Algae on drinking water.
- Pasture and crop plants as millet grass, weeds.

Chemicals as
- Carot tetrachloride.
- Corticosteroids.

Infectious as leptospirosis.
**Congenitally defective hepatic function.**
Such as inherited Southdown lambs, which has an inherited defect in excretion of bile.

**Clinical signs**
- It may be accompanied with photosensitive dermatitis.
- Skin lesions are characteristic and concentrated on dorsum side of back, vulva, lateral aspect of teats face ear.
- Lesions are markedly clear from the normal skin.
- Erythema edema of the skin.
- Intense irritation, lacerating face and may be rubbing it.
- When the teats are affected, kicking at the belly and swimming into ponds to cool areas of pain.
- Dyspnoea due to nasal obstruction, Dysphagia due to swelling of lips and closure of eyelids.
- The skin lesions may be extensive and cause shock.
- Nervous manifestation blindness, posterior paralysis.
- Peculiar sensitivity to water may seen in sheep with facial eczema.

**Diagnosis**
- Primary cause must be detected.
- Differential diagnosis from mycotic dermatitis and clostridium novyi in sheep.
- The uses of serum enzyme tests are recommended.

**Treatment**
**A) General treatment includes**
1- Immediate removal from direct sunlight.
2- Laxatives to eliminate toxic materials.
3- Prevent further ingestion of toxic substance.

**B) Local treatment includes**
1- Antihistaminic, corticosteroids.
2- Prophylactic dose of antibiotics.
3- Local application of ointments-Astringents.

**SEBORRHEA**
It is an excessive excretion of sebum on the skin surface.

**Etiology**
- True of primary seborrhea mainly occurs in human is rarely in animals.
- Secondary seborrhea accompanied with dermatitis and skin irritation as in eczema, mange and in Cases of: -
  1- Greasy heel in horse.
  2- Flexural seborrhea in cattle.
  3- Exudative epidermitis in pigs.

**Pathogenesis:**
Increased blood supplies to the skin and increase hair growth will leads to increase excretion of sebum.
Clinical findings

- Primary seborrhea
  - Greasiness of skin with oily substance of sebum.
  - Trophy of sebaceous gland may be observed on histology.

- Secondary seborrhea
  - a. Flexural seborrhea, lesions found on groins and medial aspect of thighs, fissure between the two halves of udder. Extensive outpouring of sebum, malodorous with irritation is seen. Shedding of oily skin leaving raw area under the lesion.
  - b. Greasy heel in horses
    - Usually in hind legs due to prolonged standing in unsanitary and muddy barns.
    - Thickening at the part of lesion at the coronet and painful to touch causing lameness.
    - Soreness and excoriation called scratches at the pastern and may extend to coronary band.
    - When the thickening of skin and subcutaneous tissues is marked, it will interfere with normal movement of the limbs.

Diagnosis

- It must be defined whether the lesions are primary or secondary.
- Flexural seborrhea may be mistaken with injury.
- Greasy heel seborrhea may be mistaken with chorioptic mange.

Treatment

1- Treat original cause.
2- Antibiotics and antiparasitic.
3- Washing with warm water and soap and keep the skin at the affected part clean and dry.
4- Application of an ointment locally made up of:
   - 5 part salicylic acid
   - 3 part boric acid
   - 2 part phenol
   - 2 part mineral oil
   - 2 part petrol jelly
   At 5- days interval will result good prognosis and treatment in greasy heel of horse.
5- Moving the animal to dry and clean land.

(10) ACHROMOTRICHA

It is bands of depigmentation in black wool fleece due to transient deficiency of copper in diet. In cattle deficiency of copper and increase molybdenum in diet will result in depigmentation of skin.

Causes

1- Copper deficiency and increased molybdenum in diet.
2- Damaged or destroyed pigment cells “melanocytes” by the pressure as harness, Parafilaria infestation.
3- Congenital vitiligo in the offspring of mares.
Symptoms
- Marked bands of depigmented coat mainly around the eyes.
- In horse, perineum, prepuce, face, under the tail, are the main involved area.
- The entire coats also change of its normal colour due to decrease concentration of melanocyte cells.

(11) ACNE
The term acne refers to all infections of hair follicles by the acne bacillus, which is diphtheria organism, but it may also include all suppurative organisms including staphylococci and more properly known as sycosis.

Etiology
- Staphylococcal dermatitis in horse.
- Canadian horse pox caused by corynebacterium pseudotuberculosis.
- Demodectic mange.

Pathogenesis
- When insspiated secretion and debris block sebaceous gland ducts or by pressure, it will predispose the condition.
- Hypertrophy of sebaceous glands and increased excretion also predispose to the condition.

Clinical findings
- Formation of nodules at the base of hair follicles and then pustules.
- It may be ruptured and contaminate the surrounding skin and further lesions appears.
- Hair at the site of lesions is usually easy to shed.
- The lesions are painful and rupture under pressure.

Clinical pathology
Swabs for bacteriological and parasitological examination are helpful

Diagnosis
Clinical signs has to be differentiated from impetigo.

Treatment
- Clean the skin and wash with disinfectants.
- Local application of antimicrobial ointment.
- Systemic administration of antibiotic in generalized course.
- Isolation of infected animals.

(12) IMPETIGO
It is a superficial eruption of thin – walled, usually small vesicles surrounded by a zone of erythema, the vesicles develop into pustules and rupture to from scabs.

Etiology
In animals the main organism is staphylococcus organism and most causes are : Udder impetigo in cows, infectious dermatitis or contagious Pyoderma of baby pigs caused by unspecified strept and staphylococci.

Pathogenesis
Causative organism enters through abrasion, causing lesions followed by rupture of vesicles and resulting in contamination of surrounding skin.

Clinical findings.
- Vesicles are seen on hairless parts of the body and surrounded by erythema.
- Rupture of the vesicles and causing scab formation.
3- Involvement of hair follicles and leads acne formation.

Treatment
1- Local treatment.
2- Bathing of animals with germicidal skin wash twice daily is usually adequate.

DISEASES OF THE SUBCUTANEOUS

1. SUBCUTANEOUS ABSCESS (BOLLS = FURUNCLES)

Most of conditions is due to traumatic penetration of skin with infection and may reach through the blood, or may be due to ingestion as that of traumatic reticuloperitonitis. It differs from acne so in subcutaneous abscess it involves the deeper layer of skin mainly due to corynebacterium in horses. The lesions appear on the pectorum of horses and on the face of cattle fed on roughage containing squirrel tail grass.

Multiple subcutaneous abscesses may occur in the course of many disease as a part of suppurative process in:
Corynebacterium pyogens and actinobacillus spp.

Treatment
* The abscesses must be opened drained and treated as open wound.
* Parental antimicrobial is ineffective.

2. SUBCUTANEOUS EDEMA (ANASARCA)

It is the accumulation of serous fluid in the tissue spaces of the subcutis and secondarily of the dermis. This occurs due to increased diffusion of the serous fluid. The term anasarca is applied when the abdomen is affected by the edema and the accumulation is extensive. Subcutaneous edema may be classified according to its cause of origin into:

(1) Non Inflammatory edema
Signs of inflammation are absent so that there is neither reddening nor pain, and the affected areas are cool so the term cold edema is applied and the common causes of such conditions are :-

* Increased vascular resistance:
This means increased hydrostatic pressure in the capillaries due to :
1- Congestive heart failure.
2- Vascular compression by tumors as in lymph sarcoma of mediastinal lymph nodes. Specially Udder engorgement in cattle.

* Hypoproteinemia
1- In liver damage with reduced albumin production due to liver insufficiency specially fascioliasis.
2- Renal damage with loss of protein into urine rarely occurs in animals.
3- Protein starvation.
4- Intestinal nematodiasis as heavy hookworm infestation.

* Vascular damage
In purpura hemorrhogica – there are extensive subcutaneous aggregation of protein-rich fluid to vascular damage.

Hypovitaminosis A. in beef cattle, and cobalt deficiency in sheep.

Angioneurotic edema due to an allergic origin and vascular damage.

Horses standing in black walnut shaving as bedding for a long period.

(2) Inflammatory Edema

1. In many bacterial diseases as:- Johne’s disease, Anthrax blackleg, malignant edema, pasteurellosis.
2. In many viral diseases as: Equine infectious anemia and African horse sickness.
- This edema occurs due to damage of small blood vessels by toxins of the infectious agents.
- Increased temperature characterizes inflammatory edema in initial stage at least. So known as “hot edema” redness in unpigmented skin and painful reactions.

Pathogenesis

The accumulation of fluids may be due either to: increased venous pressure, to decreased osmotic pressure of the blood and result in hypoproteinemia or due to damage of vessels resulting in leakage of fluid to tissue spaces.

Clinical findings

1. There is visible swelling, either local or diffuse.
2. Skin is puffy or doughy and pits on pressure.
3. Inflammatory signs may be found “in inflammatory edema”.
4. Edema in large animals is confined to ventral aspects of the trunk.

Diagnosis

1. Clinical findings.
2. Differential diagnosis as subcutaneous edema may be confused with infiltration; of the belly well with urine in case of urethral obstruction also may be confused with subcutaneous hemorrhage.

Treatment

Unless the primary condition is repaired, removal of the fluid by drainage methods such as intubations and multiple incisions, and use of diuretics will be of little value.

(3). Angioneurotic Edema

Is the sudden appearance of transient subcutaneous edema due to allergic cause?

Etiology

Endogenous or exogenous allergens, which result in either local or diffuse subcutaneous edema. This mainly occurs in horses and cattle on pasture especially when the pasture is in the flower stage; this suggests that allergen is of plant origin. Fishmeals also may provoke such condition. Recurrence in individual animals is common.

Pathogenesis

Allergen, release of histamine, local vascular dilatation and damage of capillary walls and leakage of plasma through damaged vessels Result in edema after an initial erythema.

Clinical findings

1. Usually there are general signs except in rare cases where bloat, diarrhea, or dyspnoea are manifested.
2. In local angioneurotic edema, the most common area is the head although perineum and udder maybe involved.
3. Diffuse lesions found in muzzle, eyelids and sometimes in conjunctive and cheeks. Conjunctiva and nictitating membrane are puffy and swollen; nictitating membrane may protrude with profuse lacrimation.
4- There is pain on touching of affected parts but irritation may evidenced by shaking of head and rubbing against objects.
5- Salivation and nasal discharge may be present.
6- When the perineum is involved, vulva is swollen, udder and peri-anal skin is swollen and edematous and there is some irritation in udder, which is evidenced by paddle with hind legs.
7- Limbs may be affected usually from the knee or hocks down to the coronet.

**Clinical pathology**
Eosinophils count may be increased from 4-5% to 12-15% or may be in normal range.

**Diagnosis**
- Sudden onset with sudden appearance in the predilection sites typifies this condition.
- Differential diagnosis from purpura hemorrhagica in later state the hemorrhage is obvious in mucosae.

**Treatment**
- Spontaneous recovery is the rule but treatment is often administered.
- Remove cattle from the pasture and fed on dry ration for at least one week to prevent reoccurrence.
- I/M or I/v injection with antihistaminic in 0.5-1.0 gm.
- Adrenaline in dilution 1/1000 at the dose of 5 ml also may aid in treatment.
- Purgatives may be used for hasten the elimination of exogenous allergens.

**4. CUTANEOUS FLUCTUATION**
This is production of a wave-like movement in a subcutaneous fluid – filled cavity (abscess, haematoma or cyst) that produces a rippling movement in the overlying skin. To demonstrate it, pressure is applied in an alternate manner with two fingers of one or both hands, when one finger is pressed inwards the other is lifted (the result of equal distribution of pressure in a fluid).

**5. SUBCUTANEOUS EMPHYSEMA**
It is the accumulation of free gas in the subcutaneous tissue.

**Etiology**
- Air entering through a cutaneous wound either surgically or accidentally.
- Lung puncture by the end of fractured rib.
- Internal penetrating wound as traumatic reticulitis.
- Rumen gases migrating from a rumenotomy or trocharization.
- Extension from pulmonary emphysema.
Subcuteears emphysema could be classified into: Aspiratory or exogenous emphysema and endogenous or septic emphysema.

**Clinical findings**
- The swelling is soft fluctuating and crepitant to touch.
- There is no pain and no external lesion on skin except in gas gangrene; skin discoloration, coldness and oozing of serum and general systemic disturbances might be observed.
**Diagnosis**
Clinical findings and bacteriological examination.

**Treatment**
- Treatment of primary cause.
- Sterile emphysema requires no treatment except if extensive multiple incisions may be necessary.
- Gas gangrene requires immediate and drastic treatment with antibiotics.

(6). **SUBCUTANEOUS HEMORRHAGE**
Subcutaneous hemorrhage occurs as a result of extravagation of whole blood into the subcutaneous tissues.

**Etiology**
1- Dicoumarol poisoning from moldy sweet clover hay.
2- Purpura hemorrhagica in horse.
3- Some diseases, which cause petechiation. Lesions are observed only in mucosa “granulocytopenic disease”.
4- Hemangiosarcoma are often present in subcutaneous tissues.

**Pathogenesis**
It may be due to either, defects in clotting mechanisms, or damage in capillary wall, which occurs due to allergic states such as purpura hemorrhagic.

**Clinical findings**
- Diffuse subcutaneous swelling.
- No visible lesions on the skin.

**Diagnosis**
Subcutaneous hemorrhage associated with hemorrhage in other organs. Paracentesis of swelling.

**Treatment**
- Removal of the cause is of great importance and priority.
- Lesion should not be opened until clotting is completed.
- If blood loss is severe, blood transfusion is required.
- If hemorrhage is recent, injection of blood coagulants is advisable.

(7). **GANGRENE**
Gangrene is the result of death of tissues with subsequent sloughing of the affected part, and when it occurs in the skin it always involves dermis, epidermis and subcutaneous tissues.

**Etiology**
1- Server continued trauma as pressure sores, saddle and harness galls.
3- Strong cold or heat.
4- Bacterial infections as bovine ulcerative mammitis, Staph. mastitis in sheep, Clostridial infection and Erysipelas in pigs.
5- Local vascular obstruction by thrombus or arterial spasm causing skin gangrene, poisoning by Claviceps purpura and Aspergillus.
6- In systemic infections in which bacterial emboli block the vessels e.g. Salmonellosis in calves and tail vaccination mycoplasma mycoides.
7- Final stage of photosensitive dermatitis and flexural seborrhea.
Pathogenesis
The basic cause of gangrene is interference with local blood supply to certain area due to: Compression of arteries, by swelling of skin as in photosensitization and arterial spasm or damage by bacterial toxins.

Clinical findings
1- If the arterial supply and drainage system is involved the initial lesion will be moist.
2- Area is swollen – raised, discolored and cold.
3- Demarcation between normal and affected part is obvious.
4- Sloughing may occur before drying the underlying surface is raw and weeping.

If the veins and lymphatics remain patent:
1- The lesion is dry from the beginning.
2- Area is cold, discolored and sunken.
3- Sloughing occur and the underlying surface usually consists of granulation tissue.

Treatment
Local application of astringent and antibacterial ointment to facilitate separation of gangrenous tissue and to prevent bacterial infection, treatment of primary condition.