Diseases of the Hair, Wool, Follicles, and Skin Glands

Alopecia And Hypotrichosis

Etiology

Alopecia and hypotrichosis are defined as lack of hair in any quantity on a normally haired body surface.

In contrast to **alopecia,** which describes hair loss of a skin surface with previously normal hair growth.

Hypotrichosis refers to a condition where there was no hair growth or abnormally low hair growth in the first place.

Both may be caused by the following conditions.

1- **Failure of Follicles to Develop**

• Congenital hypotrichosis

• Hypotrichosis in piglets without dental dysplasia

2- **Loss of Follicles**

• Cicatricial alopecia as a result of scarring after deep skin wounds that destroy follicle.

3- **Failure of the Follicle to Produce a Fiber**

a- Congenital such as Inherited symmetric alopecia and Congenital hypotrichosis**.**

b- Acquired such as Neurogenic alopecia as a result of peripheral nerve damage and Infection in the follicle.

4- **Loss of Preformed Fibers**

a- Dermatomycoses—ringworm

b- Mycotic dermatitis in all species.

c- Metabolic alopecia subsequent to a period of malnutrition or severe illness (e.g., calves having suffered severe diarrhea)

d- Alopecia of calves fed milk replacer containing fats of nonanimal origin (whale, palm, or soya oil).

e- Traumatic alopecia as a result of excessive scratching or rubbing associated with louse, tick, or itch-mite infestations; rubbing against.

f- Poisoning by thallium, selenium, arsenic and mercury.

Pathogenesis

1- In inherited hair defects the underlying cause can be disturbed hair follicle formation resulting in a reduced hair follicle quantity or disturbed functionality of hair follicles that are present in adequate numbers.

2- Noncicatricial alopecia is caused by reversible trauma to previously functional hair follicles by inflammation or mechanical trauma, which results in disturbed or interrupted synthesis in the hair bulb and ensuing shedding or fracture of hairs.

3- Cicatricial alopecia is characterized by an irreversible destruction of hair follicles most commonly caused by physical, chemical, or thermal injury or severe inflammation.

4- Chemical depilation produced by cytotoxic agents, such as cyclophosphamide, occurs as a result of induced cytoplasmic degeneration in some of the germinative cells of the bulb of the wool follicle. The alteration in cell function is temporary, so that regrowth of the fiber should follow.

Clinical Findings

1- When alopecia is a result of breakage of the fiber, the stumps of old fibers or developing new ones may be seen.
2- When fibers fail to grow, the skin is shiny and in most cases is thinner than normal.
3- In cases of congenital follicular aplasia, the ordinary covering hairs are absent.
4- Absence of the hair coat makes the animal more susceptible to the effects of sudden changes of environmental temperature.
5- There may be manifestations of a primary disease and evidence of scratching or rubbing.

Clinical Pathology

If the cause of the alopecia is not apparent after the examination of skin scrapings or swabs, a skin biopsy will reveal the status of the follicular epithelium.

TREATMENT

Primary treatment consists of removing the causes of trauma or other damage to fibers.

In cases of faulty follicle or fiber development treatment is not usually attempted.

Achromotrichia

Achromotrichia is a deficient pigmentation in hair or wool fiber, which may manifest as follows:

1- Bands of depigmentation in an otherwise black wool fleece are the result of a transitory deficiency of copper in the diet.
2- Cattle on diets containing excess molybdenum and deficient copper show a peculiar speckling of the coat caused by an absence of pigment in a proportion of hair fibers. The speckling is often most marked around the eyes, giving the animal the appearance of **wearing spectacles**.

Leukoderma And Leukotrichia

Several skin diseases of the horse are characterized by an acquired loss of melanin pigment in the epidermis or hair. Melanocytes in the epidermis and those in the hair bulbs are frequently affected independently.

Leukotrichia occurs when the melanocytes in the hair bulbs lose their normal amount of melanin pigment. When the melanocytes

in the epidermis are affected and the skin loses normal pigmentation, the abnormality is **leukoderma**.

Whereas leukotrichia can be observed as a single entity, leukoderma

is most commonly associated with leukotrichia.

Etiology

The etiology and pathogenesis of leukoderma are unknown, but trauma, inflammation, autoimmune reactions against melanocytes, local injections with epinephrine- containing local anesthetics, and defects of the autonomous nervous system.

Seborrhea

Etiology

The etiology of seborrhea is still not understood. Historically seborrhea was considered to be the result of excessive secretion of sebum onto the skin surface.

In large animals it is always secondary to dermatitis or other skin irritations that result in excessive crusting, scaling, or oiliness, such as the following:

1- Greasy heel of horses, including infection with *S. hyicus*
2- Greasy heel of cattle.
3- Flexural seborrhea of cattle
4- Besnoitiosis of cattle associated with *Besnoitia besnoiti*

CLINICAL FINDINGS

In primary seborrhea there are no lesions, only excessive greasiness of the skin. The sebum may be spread over the body surface like a film of oil or be dried into crusts, which can be removed easily. Sebaceous glands may be hypertrophied.

Flexural Seborrhea

1- ***Etiology***: Flexural seborrhea is most common in young periparturient dairy cows.
2- ***Clinical signs:*** Severe inflammation and a profuse outpouring of sebum appear in the groin between the udder and the medial surface of the thigh or in the median fissure between the two halves of the udder.
3- Extensive skin necrosis follows, causing a pronounced odor of decay, which may be the first sign observed by the owner.

4- Irritation may cause lameness, and the cow may attempt to lick the part. Shedding of the oily, malodorous skin leaves a raw surface beneath; healing follows in 3 to 4 weeks.

Greasy Heel of Cows

1- ***Etiology***: Cows grazing constantly irrigated, wet pastures or in very muddy conditions in tropical areas.
2- ***Clinical signs***: May develop local swelling, with deep fissuring of the skin and an outpouring of vile-smelling exudate on the back of the pastern of all four feet but most severely in the hind limbs.
3- Affected animals are badly lame, and their milk yield declines sharply.
4- ***Treatment***: Moving the cows to dry land and treating systemically with a broad-spectrum antibiotic effects a rapid recovery.

Greasy Heel of Horses (Scratches)

1- ***Etiology***: Greasy heel occurs mostly on the hind pasterns of horses that stand continuously in wet, unsanitary stables. It has been suggested that secondary infections associatedwith either *S. aureus* and *D. congolensis* may be causative factors. Dermatophytosis, chorioptic mange, and photosensitization are also possible causative factors.
2- ***Clinical signs***: Lameness and soreness to touch are a result of excoriations called scratches on the back of the pastern that extend down to the coronary band.
3- The skin is thick and greasy; if neglected, the condition spreads around to the front and up the back of the leg. This involvement can be severe enough to interfere with normal movement of the limb.

Clinical Pathology

The diagnosis is based on the clinical presentation and on ruling out other skin conditions resulting in abnormal cornification and keratinization.

Treatment

1- With secondary seborrhea the primary objective of treatment must be to resolve the underlying cause.
2- Topical and symptomatic treatment of the affected skin.
3- Seborrheic shampoos and lotions can either be keratolytic or keratoplastic.
4- Emollients are useful after washing the skin to rehydrate, lubricate, and soften the skin.
5- In severe cases associated with pyoderma or even skin necrosis, the use of local and systemic broad-spectrum antibiotics may be indicated.

(ANASARCA)

ETIOLOGY

Extensive accumulation of edema fluid in the subcutaneous tissue is part of general edema and is caused by the same diseases, as follows.

A-Increased Hydrostatic Pressure

1- Congestive heart failure
2- Vascular compression by a mass (e.g., anterior mediastinal lymphosarcoma, large hematoma)
3- Vascular obstruction of blood vessels or lymphatic vessels (e.g., thrombophlebitis or thrombosis)

B-Hypoproteinemic (Hypooncotic) Edema

1- Reduced albumin production in the liver associated with chronic inflammation or liver insufficiency (e.g., fascioliasis or liver cirrhosis)
2- Nephrotic syndrome with protein loss into urine (e.g., renal amyloidosis in cattle)
3- Protein-losing enteropathy (e.g., intestinal nematodiasis or paratuberculosis in cattle)

C-Increased Blood Vessel Permeability

1- Inflammation (e.g., **dourine** of horses or equine infectious anemia, bacterial infections by *Clostridium* spp. Or *Anthrax*)
2- Allergic reaction (e.g., purpura hemorrhagica of horses, insect stings)

D-Fetal Anasarca

1- Sporadic cases resulting from unknown causes are sometimes associated with deformities (e.g., in Awassi sheep).
2- Congenital absence of lymph nodes and some lymph channels causes edema to be present at birth.

PATHOGENESIS

Alteration in the balance between the hydrostatic pressure of intravascular fluids, the blood and lymph, and the osmotic pressure of those fluids or changes in the integrity of the filtering mechanism of the capillary endothelium (leaky vessels) leads to a positive advantage by the hydrostatic pressure of the system and causes a flow of fluid out of the vessels into the tissues.

Clinical Findings

1- There is visible swelling, either local or diffuse.
2- The skin is puffy and pits on pressure.
3- There is no pain unless inflammation is also present.
4- In large animals the edema is usually confined to the ventral aspects of the head, neck, and trunk and is seldom seen on the limbs.

Clinical Pathology

Differentiation between obstructive and inflammatory edema can be made by cytologic and bacteriologic examination of the fluid.

Treatment

1- Primary treatment requires correction of the primary causal abnormality.
2- Supportive treatment will also depend on the underlying cause but can consist of transfusing plasma or whole blood in cases of hypooncotic edema, or antiinflammatory or diuretic therapy in cases of inflammatory or allergic edema.

ANGIOEDEMA
(ANGIONEUROTIC EDEMA)

Transient, localized subcutaneous edema as a result of an allergic reaction and caused by endogenous and exogenous allergens provokes either local or diffuse lesions.

Angioedema occurs most frequently in cattle and horses on pasture, especially during the period when the pasture is in flower.

Etiology: This suggests that the allergen is a plant protein. Fish meal may also provoke an attack

Angioedema can also occur as adverse reaction to parenteral administration of certain antibiotics, vaccines, blood, plasma, or other IV fluids.

Pathogenesis

Most cases appear to be associated with a type I or type III hypersensitivity reaction. After an initial erythema, local vascular dilatation is followed by leakage of plasma through damaged vessels.

CLINICAL FINDINGS

A-Local lesions

1- Most commonly affect the head, with diffuse edema of the muzzle, eyelids, conjunctiva, and cheeks.
2- Affected parts are not painful to touch, but shaking the head and rubbing against objects suggest irritation.
3- Salivation and nasal discharge may be accompanying signs.

B-Perineal involvement includes

1- Vulvar swelling, often asymmetric, and the perianal skin, and sometimes the skin of the udder.
2- Edema of the lower limbs, usually from the knees or hocks down to the coronets, is a rare sign.

C- Systemic signs are absent.

CLINICAL PATHOLOGY

The blood eosinophil count is often within the normal range, but may be elevated from a normal level of 4% to 5% up to 12% to 15%.

DIFFERENTIAL DIAGNOSIS

Diagnostic confirmation is found with sudden onset and disappearance of edema at the typical sites.

A- Subcutaneous edema as a result of vascular pressure occurs mostly in dependent parts and is not irritating.

In horses, and rarely in cattle, angioedema may be simulated by **purpura hemorrhagica**, but hemorrhages are usually visible in the mucosae in purpura.

TREATMENT

Acute anaphylaxis with angioedema:

1- Epinephrine: 3 to 5 mL/ 450 kg of a 1 : 1000 solution IM or SC (can be combined with steroids) (R-1)
2- Acute angioedema in horses:
   a- Dexamethasone soluble 0.01 to 0.1 mg/kg IV or IM q24 h for 3 to 7 days (R-1)
   b- Hydroxyzine hydrochloride 0.5 to 1.0 mg/ kg IM or PO q8 h (R-2)
   c- Diphenhydramine hydrochloride 0.7 to 1 mg/kg q12 h (R-2)
   d- Chlorpheniramine 0.25 to 0.5 mg/kg q12 h (R-2).

SUBCUTANEOUS EMPHYSEMA

ETIOLOGY

Emphysema, free gas in the subcutaneous tissue, occurs when air or gas accumulates in the subcutaneous tissue as a result of the following:

1- Air entering through a cutaneous wound made surgically or accidentally, particularly in the axilla or inguinalregion
2- Extension from pulmonary emphysema
3- Air entering tissues through a discontinuity in the respiratory tract lining (e.g., in fracture of nasal bones; trauma to pharyngeal, laryngeal, and lung puncture by a fractured rib; trauma to the trachea during an attempt to pass a nasoesophageal tube
4- Extension from vaginal lacerations in cattle, particularly in cattle with vaginal prolapse and following dystocia.
5- Gases migrating from abdominal surgery because the abdominal cavity is usually at a negative pressure relative toatmospheric pressure.
6- Gas gangrene infection

PATHOGENESIS

Air moves very quickly in a dorsal manner through fascial planes, especially when there is local muscular movement.

CLINICAL FINDINGS

1- Visible subcutaneous swellings are soft, painless, fluctuating, and grossly crepitated to the ctouch, but there is no external skin lesion.
2- In gas gangrene, discoloration, coldness, and oozing of serum may be evident. Affected areas of skin are moderately painful to touch.
3- Emphysema may be sufficiently severe and widespread to cause stiffness of the gait and interference with feeding and respiration.

DIFFERENTIAL DIAGNOSIS

Diagnostic confirmation is based on the observation of crepitus and the extreme mobility of the swelling; these distinguish emphysema from other superficial swellings.

1- **Anasarca**, dependent and pits on pressure.
2- **Hematoma, seroma at injury sites**, confirmed by needle puncture.
3- **Cellulitis** is accompanied by toxemia, confirmed by needle puncture.

TREATMENT

Primary treatment is to address the source of the air, but this may be impossible to locate or to close.

Supportive treatment is only necessary in the extremely rare case where emphysema is extensive and incapacitating, in which case multiple skin incisions may be necessary. Gas gangrene requires immediate and drastic treatment with antibiotics.

LYMPHANGITIS

Lymphangitis is characterized by inflammation and enlargement of the lymph vessels and is usually associated with lymphadenitis.

Etiology

Common causes are as follows.

Cattle

1- Bovine farcy caused by *Mycobacterium farcinogenes*
2- Cutaneous tuberculosis associated with atypical mycobacteria, rarely *Mycobacterium bovis*

Horse

1- Epizootic lymphangitis (equine histoplasmosis) as a result of *Histoplasma capsulatum var. farciminosum.*
2- Ulcerative lymphangitis as a result of *Corynebacterium pseudotuberculosis*
3- Glanders (farcy) caused by *Burkolderia mallei*
4- Sporotrichosis
5- Sporadic lymphangitis
6- Strangles in cases where bizarre location sites occur
7- In foals, ulcerative lymphangitis associated with *Streptococcus zooepidemicus*.

Pathogenesis

Spread of infection along the lymphatic vessels causes chronic inflammation and thickening of the vessel walls. Abscesses often develop, with discharge to the skin surface through sinuses.

Clinical Findings

1- An indolent ulcer usually exists at the original site of infection.
2- The lymph vessels leaving this ulcer are enlarged, thickened, and tortuous.
3- Local edema may result from lymphatic obstruction.

In chronic cases much fibrous tissue may be laid down in the subcutis, and chronic thickening of the skin may follow.

Clinical Pathology

Bacteriologic examination of discharge for the presence of the specific bacteria or fungi is common practice.

TREATMENT

Primary treatment requires vigorous, early surgical excision or specific antibiotic therapy.

Supportive treatment is directed toward removal of fluid and inflammatory exudate and relief of pain.

HEMATOMA

Hematoma refers to extravasation of whole blood into the subcutaneous tissues.

Etiology

Common causes include the following:

1- Traumatic rupture of large blood vessel
2- Dicoumarol poisoning from moldy sweet clover hay
3- Purpura hemorrhagica in horses
4- Bracken poisoning in cattle; other granulocytopenic diseases manifested principally by petechiation, with lesions observed only in mucosae
5- Systemic disease associated with disseminated intravascular coagulopathy (DIC)
6- Hemangiosarcoma in subcutaneous sites
7- Neonatal bovine pancytopenia
8- Inherited hemophilia

Pathogenesis

Leakage of blood from the vascular system can cause local swellings, which interfere with normal bodily functions but are rarely sufficiently extensive to cause signs of anemia.

Clinical Findings

1- Subcutaneous swellings resulting from hemorrhage are diffuse and soft.
2- Specific locations of subcutaneous hemorrhages in horses include the frontal aspect of the chest—as a result of fracture of the first rib in collisions at full gallop, and often fatal through internal hemorrhage— and perivaginal at foaling, causing massive swelling of the perineum and medial aspect of the thigh.

Clinical Pathology

Visual examination of a needle aspirate confirms the existence of subcutaneous hemorrhage.

Diagnosis of the primary cause is greatly assisted by platelet counts and prothrombin, clotting, and bleeding times.

Treatment

Primary Treatment

Primary treatment targets removal or correction of the cause.

Supportive Treatment

The hematoma should not be opened until clotting is completed, except in the case of a massive hemorrhage that is interfering with respiration, defecation, or urination.

If blood loss is severe, blood transfusions may be required.

NECROSIS AND GANGRENE

Necrosis is tissue death; gangrene is sloughing of dead tissue. When either change occurs in the skin, it involves the dermis, epidermis, and subcutaneous tissue.

Different types of gangrene arerecognized:

A- Dry gangrene is primarily caused by arterial occlusion resulting in tissue ischemia.

Signs: Affected tissue appears dry and shrunken, with dark discoloration and a clear demarcation line from healthy tissue. There is no bacterial infection or putrefaction because bacteria fail to survive in the desiccated tissue.

B- Wet gangrene is most common after sudden blockage of venous blood flow resulting in ischemia while the affected tissue is saturated with stagnant blood.

Tissue trauma (e.g., from mechanical trauma or burns) and ischemia result in release of tissue water and give the affected area a moist and swollen appearance.

Because the moist and protein-rich tissue facilitates bacterial growth, infection with saprogenic microorganisms is common.

Signs: This infection results in the putrid and rotten aspect and odor of the tissue and may cause septicemia.

C- Gas gangrene is caused by *C. perfringens* (see also "Malignant Edema").

DIFFERENTIAL DIAGNOSIS

Confirmation of the diagnosis is by visual recognition.

1- Gangrenous mastitis in cows or ewes.
2- Photosensitive dermatitis.

TREATMENT

Primary treatment requires removal of the etiologic insult.

Supportive treatment comprising the application of astringent and antibacterial ointments may be required in cases of wet gangrene to facilitate separation of the gangrenous tissue and to prevent bacterial infection. Aggressive tissue debridement of necrotic tissue and in severe cases amputation of affected body parts may be required.