**Impetigo**

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

In humans, impetigo is specifically a streptococcal infection but lesions are often invaded secondarily by staphylococci. In animals the main organism found is usually a staphylococcus. The causative organism appears to gain entry through minor abrasions, with spread resulting from rupture of lesions causing contamination of surrounding skin and the development of secondary lesions. Spread from animal to animal occurs readily. The only specific examples of impetigo in cows are: Udder impetigo of cows

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.

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

**Differential diagnosis**

• Cowpox, in which the lesions occur almost exclusively on the teats and pass through the characteristic stages of pox

• Pseudocowpox, in which lesions are characteristic and also restricted in occurrence to the teats.

**Treatment**

1-Primary treatment with antibiotic topically is usually all that is required

because individual lesions heal so rapidly.

2- Supportive treatment is aimed at preventing the occurrence of secondary lesions and spread of the disease to other animals. Twice daily bathing with an Diseases of the epidermis and dermis efficient germicidal skin wash is usually adequate.

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**URTICARIA**

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

**ETIOLOGY:**Primary urticaria results directly from the effect of the pathogen, e.g.:

1-Insect stings

2-Contact with stinging plants

3-Ingestion of unusual food, with the allergen, usually a protein

4-Occasionally an unusual feed item, e.g. garlic to a horse

5-After a recent change of diet

6-Administration of a particular drug, e.g. penicillin; or anesthetic agent

7-Allergic reaction in cattle 8 days following vaccination for foot-and mouth diseases

8-Death of warble fly larvae in tissue

9-Milk allergy when Jersey cows are dried off

10-Transfusion reaction.

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**

The lesions are characteristic of an allergic reaction. There is degranulation of mast cells followed by 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.

Only the dermis, and sometimes the epidermis, is involved. In extreme cases the wheals may expand to become seromas, 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 FINDINGS**

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. They vary from 0.5-5 cm in diameter, with a flat top, and are tense to the touch. There is often no itching, except with plant or insect stings, nor discontinuity of the epithelial surface, exudation or weeping. Pallor of the skin in wheals can be observed only in unpigmented skin.

Other allergic phenomena, including diarrhea and slight fever, may accompany the eruption. The onset of the lesions is acute to peracute with the wheals developing within minutes to hours after exposure to the triggering agent. When associated with severe adverse systemic responses, including apnea, respiratory arrest, atrial fibrillation, cardiac arrest or sudden death, the case qualifies as one of anaphylaxis.

Subsidence of the wheals within 24-48 hours is usual but they may persist for 3-4 days because of the appearance of fresh lesions. Urticaria lasting 8 weeks or longer is classified as chronic or recurrent Urticaria, which may require testing for atopic disease using intradermal skin testing and serum testing for antigen-specific IgE.

Adverse reactions in dairy cattle following annual vaccination for foot and- mouth disease are characterized by wheals (3-20 mm in diameter) covering most of the body, followed by exudative and necrotic dermatitis. The affected areas become hairless and the wheals exude serum and become scabbed over. 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. Loss of body weight and lymphadenopathy also occur. Pruritis, depression and a drop in milk yield are common.

**CLINICAL PATHOLOGY (Diagnosis)**

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. The duration of the reaction also varies a great deal. Intradermal tests in horses without atopy and horses with atopic dermatitis or recurrent urticaria using environmental allergens indicate a greater number of positive reactions for intradermal tests in horses with atopic dermatitis or recurrent urticaria, compared with horses without atopy. This provides evidence of type-1 IgE-mediated hypersensitivity for these diseases.

Biopsies show that tissue histamine levels are increased and there is a local accumulation of eosinophils. Blood histamine levels and eosinophil counts may also show transient elevation.

**Differential diagnosis**

The differential diagnosis list is limited to angioedema, but in urticaria the lesions can be palpated in the skin itself. Angioedema involves the subcutaneous tissue rather than the skin and the lesions are much larger and more diffuse. The two conditions may appear in the one animal at the one time.

**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 treatment, especially in the relief of the pruritus.
* The local application of cooling astringent lotions such as calamine or white lotion or a dilute solution of sodium bicarbonate is favored.
* 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 2 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.

**DERMATITIS AND DERMATOSIS**

**Etiology**

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

**All species**

* Mycotic dermatitis due to *Dermatophilus congolens* , in horses,cattle, sheep
* *Staphylococcus aureus* is a common finding in cases in all species, either as a sole pathogen or combined with other agents
* Ringworm
* Photosensitive dermatitis
* Chemical irritation (contact dermatitis) topically
* Arsenic - systemic poisoning
* Mange mite infestation - sarcoptic, psoroptic, chorioptic, demodectic mange
* Strongyloides (Pelodera) sp. dermatitis

**Cattle**

* Udder impetigo - *S. aureus*
* Cowpox
* Ulcerative mammillitis - udder and teats only
* Lumpy skin disease
* Foot-and-mouth disease – vesicles around natural orifices; vesicular

stomatitis with lesions on teats and coronet.

* Rinderpest, bovine virus diarrhea, bovine malignant catarrh, bluetongue- erosive lesions around natural orifices, eyes, coronets
* Perianal vesicular and necrotic dermatitis associated with mushroom poisoning
* Dermatitis on legs - potato poisoning, topical application of irritants or defatting agents.
* Bovine exfoliative dermatitis.
* Slurry heel.

**Sheep and goats**

* Strawberry foot rot – Dermatophilus
* Sheep pox
* Contagious ecthyma
* Ulcerative dermatosis
* Rinderpest. peste de petits ruminants, bluetongue
* Foot and mouth disease and vesicular stomatitis
* Fleece rot - constant wetting and associated with P aeruginosa
* Lumpy wool ; D. congolensis
* Itch-mite infestation
* Blowfly infestation (cutaneous myiasis)
* Louse infestation.
* Ovine atopic dermatitis
* Caprine idiopathic dermatitis
* Post dipping necrotic dermatitis

**Horses**

* Staphylococcus hyicus in a syndrome reminiscent of greasy heel
* Actinomyces viscosum
* Horse pox
* Viral papular dermatitis
* Vesicular stomatitis - vesicles around natural orifices
* Sporotrichosis
* Dermatophytes, including ringworm.
* Atopic dermatitis (IgE-mediated hypersensitivity)
* Chronic eosinophilic dermatitis
* dermatitis and stomatitis.
* Cutaneous habronemiasis
* Ulcerative dermatitis, thrombocytopenia and neutropenia in

neonatal foals

**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.

**CLINICAL FINDINGS**

Affected skin areas first show erythema and increased warmth. The subsequent stages vary according to the type and severity of the causative agent. There may be development of discrete vesicular lesions or diffuse weeping. Edema of the skin and subcutaneous tissues may occur in severe cases. The next stage may be the healing stage of scab formation or, if

the injury is more severe, there may be necrosis or even gangrene of the affected skin area. Spread of infection to subcutaneous tissues may result in a diffuse cellulitis or phlegmonous lesion. A distinctive suppurative lesion is usually classified as pyoderma. Deep lesions which cause damage to dermal collagen may cause focal scarring and idiopathic fibrosing dermatitis.

A systemic reaction is likely to occur when the affected skin area is extensive. Shock, with peripheral circulatory failure, may be present in the early stages. Toxemia, due to absorption of tissue breakdown products, or septicemia due to invasion via unprotected tissues, may occur in the later stages.