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Equine pastern dermatitis, an unsolved problem?
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Introduction
The term ‘equine pastern dermatitis’ is a collective name for a variety of clinical presentations of lower limb dermatoses (colloquial names ‘scratches’, ‘cracked heels’, ‘greasy heel’, ‘dew poisoning’, ‘mud rash’ and ‘mud fever’). To establish a diagnosis, a thorough history is vitally important since the syndrome can be significantly (and often adversely) affected by prior treatments (often involving topical ‘home remedies’). Although there is no new ‘wonder treatment’, more specific therapies can be applied as a result of an improved understanding of the underlying aetopathogenesis.

Helpful laboratory procedures include skin scrapings for ectoparasites and fungi, fungal and bacterial cultures (including antibacterial sensitivity tests), and skin biopsies.

Possible causes include:
- Chorioptes equi infestation
- Fungal infections
- Superficial and deep bacterial infection
- Immune-mediated problems
- Photosensitisation

Choriopptic mange
Chorioptes equi is an easily identified, non-burrowing mite that feeds on skin debris, most often in winter in horses with feathered legs. Transmission between horses is by direct and indirect contact; asymptomatic carrier horses may harbour the infection from season to season.

Clinical signs are usually limited to the distal limbs and include pruritus, scaling and fine papular eruptions; heavy infestations can affect the whole body leading to generalised pruritus.

Fungal infections
Fungal infection restricted to the lower limbs is very rare although dermatophytosis is the most common skin infection in horses. Dermatophyte infection requires skin trauma such as minor wounds, moisture or pressure/rubbing. In some cases however, the horse may show a stronger inflammatory response that suggests an allergic or irritational process which results in pruritus.

Superficial or deep bacterial infections
Maceration of skin by water, sweat, friction from skin folds, topical treatments, abrasions, cuts, punctures, insect bites, scratching or a foreign body can all impair local cutaneous defences and thereby facilitate infection.

Folliculitis is an inflammation of the hair follicles. Disruption of the hair follicle results in a more widespread infection in the dermis and subcutis, called furunculosis. Staphylococcus aureus or, occasionally, S. intermedius are most commonly responsible. Streptococcus equi, S. zooepidemicus and Dermatophilus congolensis, and rarely Corynebacterium pseudotuberculosis, may also be responsible.

Environmental and individual hygiene form the mainstays of treatment. In mild cases anti-septic shampoos, e.g. povidone-iodine shampoo, can be effective but in serious cases a systemic antimicrobial therapy is indicated. Oral trimethoprim sulfa-combinations are safe, convenient and effective in most cases. Penicillin may be sufficient if a streptococcus is involved.

Sometimes, there is a ‘vicious cycle’ of irritation and inflammation. Then short-acting corticosteroids administered between 08.00 and 09.00 h such as prednisolone (1–2 mg/kg bwt per os q. 24 h) or dexamethasone (0.04 mg/kg bwt i.v. q. 24 h) can be justified together with the antimicrobials.

Local antimicrobial ointments may be indicated in the early stages but often these will not penetrate sufficiently to be effective. Ointment containing corticosteroids may make the skin thin and crusty with decreased hair formation.

Immune-mediated problems
Equine sarcoidosis
Although equine sarcoidosis is mainly described as a systemic disease associated with exfoliative dermatitis, severe wasting and sarcoidal granulomatous inflammation of multiple organ systems, we often encounter cases of a sarcoidosis-like dermatitis where only one or 2 lower limbs are involved. Both pigmented and nonpigmented limbs show scaling, crusting and a thinner hair coat. These horses are not systemically ill, and may perform well. If untreated the dermatitis deteriorates, the crusting gets more severe and the horse often becomes lame. The diagnosis is confirmed by biopsy. The prognosis for full recovery is poor, but 2–3 weeks of dexamethasone (0.04–0.08 mg/kg bwt q. 24 h am) i.m. or i.v. followed by low dose prednisolone (0.25–1.0 mg/kg bwt per os q. 24 h am) can be effective enough to allow these horses to remain useable. Local treatment is contraindicated as the skin is already very fragile and sensitive.

Vasculitis of the lower limb
A broad spectrum of disorders can be associated with histological evidence of inflammatory changes in the walls of blood vessels. The aetiology of most cutaneous vasculitides is not known. In the generalised vasculitis cases (mostly purpura haemorrhagica), the inciting cause is thought to be bacterial (e.g. strangles) or viral.

Pastern leucocytoclastic vasculitis or ‘photo-aggravated vasculitis’ is a specific clinico-pathological entity unique to the horse and usually limited to nonpigmented distal extremities, with the medial and lateral aspects of the pastern being the favoured sites. The individual lesions are usually multiple and reasonably well demarcated but are sometimes coalescent and occupy larger, less well-defined areas. Initially, erythema, oozing, crusting, erosions and superficial ulcerations develop, followed by oedema. In chronic cases the lesions usually develop a rough ‘warty’ surface. The pathogenesis is uncertain, but it is probably an immune-complex disease. There is circumstantial evidence that the disease may be related to exposure to plants (direct contact) or to nutritional challenges. Management consists of oral or parenteral corticosteroids and protection from light by careful bandaging or simply by avoiding sunlight exposure.

Photosensitisation
All types of photosensitisation have 3 features in common: presence of a photodynamic agent within the skin, exposure to UV light and cutaneous absorption of UV light, which is greatly facilitated by lack of pigment and hair coat. Photosensitisation is different from simple sunburn.
Photosensitisation is usually classified according to the source of the photodynamic agent:

1. Primary photosensitisation in which a preformed or metabolically derived photodynamic agent reaches the skin by ingestion, injection or contact.
2. Hepatogenous photosensitisation, where there is excessive circulating phylloerythrin as result of advanced liver disease.
3. Idiopathic photosensitisation (of uncertain or unexplained aetiology).

The dermatological presentations are essentially identical, regardless of the cause; lesions are restricted to white haired or pink skinned areas. An acute onset of erythema, oedema and variable degrees of pain with vesicles and bullae progresses rapidly to oozing, necrosis, sloughing and ulceration.

Treatment must involve removal of the photodynamic agent (and any underlying primary disease such as liver failure if possible) and avoidance of UV exposure. Symptomatic skin treatment with local soothing ointments, steroid creams and systemic nonsteroidal anti-inflammatory drugs are indicated. Systemic antimicrobials may be required.

In general the prognosis is favourable for primary photosensitisation, but very poor for hepatogenous photosensitisation.

Conclusions

Equine pastern dermatitis is often a collective name for abnormalities of the skin of the pastern region. Many of the differential diagnoses are readily recognisable from their clinical and historical features and focused treatment will usually bring about a resolution.