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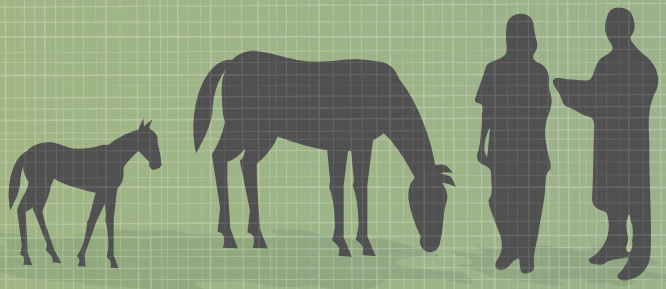

DERMATOLOGY

Chair: Edd Knowles

9.00

Diagnostic investigation of urticaria

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Urticaria (hives) is a relatively common presentation in equine practice, but can present the clinician and owner with a significant diagnostic challenge. Urticaria results from degranulation of cutaneous mast cells, most commonly due to type I hypersensitivity reactions, with allergen cross-linking of IgE antibodies on the cell surface, but nonimmunological triggers, mechanical or physical mechanisms also occur. Histamine is the predominant mediator responsible for increased blood vessel permeability and leakage of fluid leading to localised tissue oedema, but other proinflammatory mediators may also be involved, and some horses with urticaria suffer variable pruritus. The pathogenesis of urticaria in the horse remains unproven, but a role for IgE-mediated reactions has been demonstrated [1]. A study of cytokine expression and inflammatory infiltrate in lesional skin suggested that T-helper 2 cytokines, eosinophils, mast cells and presumptive macrophages play a role in the pathogenesis of equine recurrent urticaria [2].

Clinical features

The clinical presentation is usually easily recognised, although lesions can take several forms, including papules, plaques, annular and linear wheals, and even large swellings in cases of angio-oedema, sometimes with serous ooze. Urticarial lesions pit on application of digital pressure, which helps differentiate from lesions of erythema multiforme and amyloidosis. Lesions occur most commonly on the neck and trunk, but can affect head and upper limbs. Individual lesions are often transient, but new lesions may develop. The course can be short-lived, or chronic and/or recurrent. It has been estimated that up to 60% of cases of equine urticaria may be single episodes, which either self-resolve or respond to appropriate symptomatic treatment. Detailed investigations into underlying trigger factors are only indicated in patients with chronic or recurrent disease lasting longer than 1–2 months.

Underlying causes

In referral practice, the most common underlying cause of chronic or recurrent urticaria is atopic dermatitis. However, the commonest hypersensitivity disorder in which urticaria may be a clinical sign is insect bite hypersensitivity. While food triggers are commonly suspected, adverse food reactions causing urticaria are rarely confirmed, with only one published case report of urticaria associated with feeding a dry garlic supplement [3] and no case series of definitively proven food allergy in the peer-reviewed literature.

Dermatographism resulting from pressure is recognised in the horse. Other nonimmunological triggers of urticaria reported anecdotally include exercise, heat and cold. A case of sweat-induced hypersensitivity manifesting as urticaria is reported [4]. Urticaria was associated with sweating, whether or not this resulted from exercise. Adverse drug reactions commonly present with urticaria or angio-oedema, with many classes of drugs and topical chemicals being implicated.

Diagnostics

A good history is important in cases of urticaria, particularly documenting age of onset, duration of disease, seasonality, management practices, drug usage (including topical products), and response to previous treatments.

Skin biopsy is rarely helpful since histopathological findings are very nonspecific, but may be helpful for rule-outs.

Restriction or elimination tests and provocative challenge can be undertaken. This may include keeping the animal fully indoors or out at pasture, changing bedding, and changing topical products used on the animal and on tack. Elimination diets using a novel food source can be difficult to achieve, but an exclusively grass-based diet of grass nuts, haylage in preference to hay, lucerne (unless previously exposed), with exclusion of all cereals and supplements, is recommended. The appropriate duration of elimination diets in the horse has not been established, but a minimum of 1 month is advised. Any improvement should be followed by dietary provocation to confirm flare of signs after re-exposure, with subsequent improvement again on return to the strict exclusion diet. Serological tests for food allergy are unreliable [5].

For cases where a clinical diagnosis of atopic dermatitis has been made, allergen-specific IgE testing is indicated. Intradermal testing is considered the gold standard, with serological tests giving poor or no correlation with intradermal tests and with each other [6]. Results must be interpreted in light of the history, but identification of implicated allergens allows avoidance measures to be employed and offers the option of allergen-specific immunotherapy (ASIT) as part of the management regime.

References

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