Skin allergies in horses – presentations and treatment types

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ABSTRACT

Horses can develop allergic reactions to a variety of causes and show clinical signs of pruritus (itching), urticaria (raised thickened skin) and alopecia (hair loss). This often causes the skin to be damaged by excoriation (self trauma).

This article illustrates the different types of allergic reaction in horses and describes the approach to investigating and treating them, illustrated with several case studies.

An allergic reaction (or “hypersensitivity”) arises when the body is sensitised to an otherwise harmless agent (antigen).

This antigen could be:

- food agents
- inhaled (such as pollen, dust mites or mould spores)
- insect bites
- direct contact (such as bedding or tack)

Once in contact with the antigen, the immune system over-reacts, with white blood cells (lymphocytes and plasma cells) producing inflammatory mediators – such as antibodies, complement and prostaglandins – causing host cell damage.

This overreaction by the host’s immune system is called a “hypersensitivity reaction”.

Hard work

Table 1. Types of hypersensitivity

Hypersensitivity reaction	Mechanism of action	Examples
Type I	Immediate reaction – antibodies (IgE) on the surface of mast cells bind antigen in a sensitised individual and degranulate to release histamine, causing inflammation.	Atopic dermatitis, food allergy, anaphylaxis.
Type II	Antibodies (IgM or IgG) and complement bind to antigens, forming immune complexes that disrupt cell membranes and activate lymphocytes, promoting inflammation and cell death.	Haemolytic disease of foals, pemphigus, drug eruptions, thrombocytopenia.
Type III	Free antibodies in the circulation bind antigens, forming immune complexes that are deposited in capillary beds, attracting other inflammatory cells, neutrophils releasing inflammatory mediators and causing cell damage.	Vasculitis, purpura haemorrhagica, systemic lupus erythematosis.
Type IV	T cell-mediated (cluster of differentiation 4 T helper cell type 1), delayed type hypersensitivity; antigens bind to tissue proteins to form an immune complex that is picked up by a specialised antigen-presenting cell (monocyte, macrophage or Langerhans cell) and transported to the lymphoreticular system where T lymphocytes react by producing inflammatory mediators, which cause tissue damage and pruritus.	Drug eruptions, insect bite reactions (sweet itch).

Table 1. Types of hypersensitivity (click to zoom).

The great difficulty with treating skin diseases is, despite the differing causes, many problems look the same.

Plus, regardless of whether the disease is parasitic, fungal, bacterial or allergic, the horse is left with itchy skin that, through rubbing and nibbling, becomes sore and hairless.

Allergic reactions can be triggered by the skin coming into contact with an allergen (contact dermatitis), an allergen being eaten (food allergy), an allergen being inhaled (allergic inhalant dermatitis, or atopy) or an allergen being injected by insect bites (insect bite hypersensitivity, such as sweet itch).

Reaction types

Four different types of hypersensitivity have been described (**Table 1**) and these immune reactions are involved to various degrees in different allergic patterns. For example, atopy and contact allergy bring on type I, food allergies bring on types I and III, while insect bite hypersensitivity (sweet itch) causes types I and IV (Pascoe and Knottenbelt, 1999).

A common presentation in horses is urticaria mediated by the antibody IgE (Rufenacht et al, 2005). However, this can be triggered by both immunological and non-immunological mechanisms (Littlewood, 1991) and may be acute (less than six to eight weeks' duration) or chronic (greater than six to eight weeks' duration; Fadok, 1990).

Unless the problem is properly investigated, it is difficult to decide on the most appropriate treatment, so less chance exists of getting the problem resolved quickly. If inappropriate treatment is administered, it may not get better and possibly may get even worse.

Integral investigation

Table 2. Contagious causes of pruritus

Bacterial	Fungal	Parasitic	Viral
Dermatophilus: rainscald/mud fever	Dermatophytosis: trichophyton, microsporum	Lice: haematopinus, damalinia	Coital exanthema
Folliculitis: cutaneous/pastern		Mites: chorioptes, harvest mites, dermanyssus	
		Helminth: *Oxyuris equi*, onchocerca	

Table 2. Contagious causes of pruritus (click to zoom).

The many causes of pruritic skin disease can be classified into contagious (**Table 2**) and non-contagious (**Table 3**), and a thorough clinical examination and diagnostic workup – with skin scrapes, hair plucks and fungal cultures or skin biopsies – is necessary to allow a diagnosis and development of an effective treatment plan (Paterson, 2000).

This approach (Sloet van Oldruitenborgh-Oosterbaan and Grinwis, 2016) involves:

- getting a thorough history
- making a clinical and dermatological examination
- preparing a list of differential diagnoses
- using laboratory tests to rule in or out differential diagnoses to make a specific diagnosis
- putting together a treatment plan
- reassessing the case to monitor the response to treatment and review the diagnosis

Table 3. Non-contagious causes of pruritus

Infectious	Physical	Immune-mediated
Bacterial: staphylococcal folliculitis	Chemical: blisters, environmental	Vasculitis Autoimmune: pemphigus, drug eruption
Parasitic: Fleas – stickfast Flies – Tabanidae, *Stomoxys, Simulium* Stinging insects – wasps/bees Onchocerca	Irritant contact dermatitis: feed/ bedding/irritant chemicals – leather treatments	Hypersensitivities: atopy, allergic contact dermatitis, insect bite – sweet itch, food

Table 3. Non-contagious causes of pruritus (click to zoom).

The VN can help the vet here by getting as much information from the owner as possible before seeing the case, using questions such as these (Diesel, 2014):

General questions

- What is the environment? Table/pasture/bedding?
- General health: appetite, thirst, droppings
- Current/recent medications

- Vaccination, worming protocol
- Fly control
- Feeding regime/diet/supplements
- Travel history
- Contact with other animals, horses
- General use: endurance/eventing/hunting
- Breeding: Arabian, American quarter horse, Welsh pony, and so on

Dermatology-specific questions

Figure 1. Microscope and equipment to investigate skin disease.

- Age of onset
- Frequency of recurrence: first episode, seasonality
- Duration of symptoms
- Trigger factors: exercise/heat/cold/environment
- Location of lesions: contact with tack/rugs
- Degree of pruritus
- Response to medication
- Contagion evidence: other horses/grooms
- Other symptoms: breathing problems – chronic obstructive pulmonary disease

The VN should also have the following appropriate equipment ready for investigating the problem (**Figure 1**):

For skin scrapes

- Microscope
- Microscope slides and cover slips
- Surgical blades
- Liquid paraffin
- Special stains
- Immersion oil

For skin biopsies

- Syringes and needles
- Drugs for sedation and local anaesthetic
- Surgical kit and blades
- Sample pots and formal saline fixative
- Suture material

For microbiology

- Sample pots to send off hair samples for fungal culture
- Swabs to send off for bacterial culture and sensitivity

For blood tests

- Vacutainers and needles

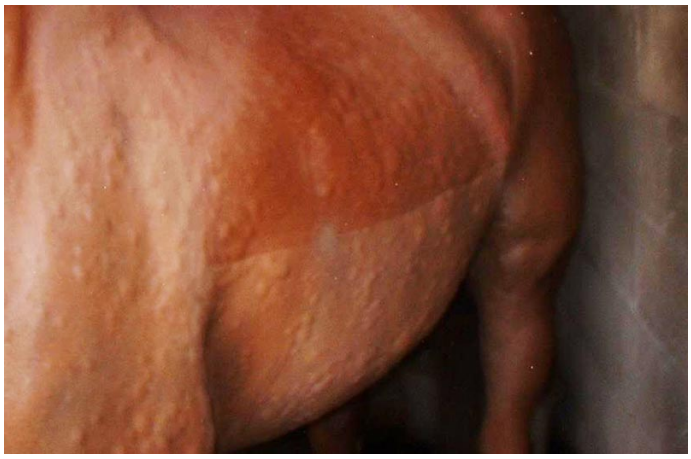

Figure 2. Contact allergy to wheat straw.

A large amount of information to help with the diagnosis can be found from taking the history. Initially, the predominant clinical sign should be identified. Commonly, in equine skin, these are categorised as follows (Paterson and Ball, 2013):

- pruritus (itching)
- alopecia (hair loss)
- nodular lesions
- crusting and scaling
- alterations in pigmentation
- ulcerations and erosions
- pedal or pastern dermatitis

Following history taking, clinical examination and recording of the owner‘s complaint, a problem list can be compiled.

During the dermatological examination, skin lesions are classified as either primary or secondary. However, some lesions can be both:

Primary lesions

An initial lesion that develops spontaneously as a direct reflection of the underlying disease.

Macule (1cm)

A circumscribed, non-palpable spot characterised by a change in skin colour (melanin, erythema or haemorrhage).

Papule (1cm)

A solid palpable elevation of the skin.

Pustule

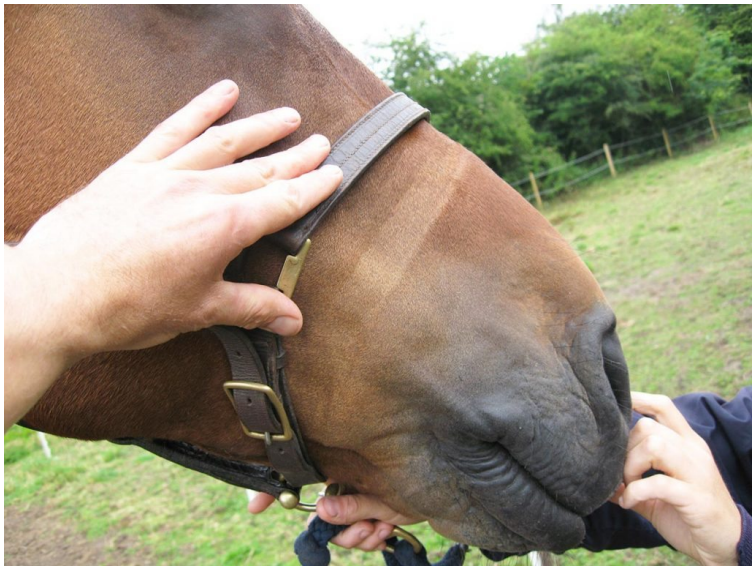

Figure 3. Urticaria and oedema of the head.

Epidermis elevation filled with pus. Location can be intradermal, sub-epidermal or follicular.

Abscess

Demarcated fluctuant lesion resulting from dermal or SC accumulation of pus not visible on the skin surface until it drains.

Vesicle (1cm)

Sharply circumscribed elevation of the epidermis filled with clear fluid (occurs with viral, autoimmune or irritant dermatitis).

Cyst

An epithelium-lined cavity containing fluid or solid material. In the skin, they are usually lined by adnexal epithelium (hair follicle, sebaceous or epitrichial cells) and are filled with cornified cellular debris, or sebaceous or epitrichial secretions.

Tumour

A large mass involving any structure of the skin or SC tissue (may be normal, neoplastic or granulomatous tissue).

Nodule

Circumscribed solid elevation >1cm extending into deeper layers of skin and is the result of a massive infiltration of inflammatory or neoplastic cells into the dermis or subcutis (deposition of fibrin or crystalline material forms calcinosis cutis).

Wheal

White/pink, sharply circumscribed, raised lesion due to oedema. Appears and disappears within minutes/hours, has no effect on the appearance of overlying skin or hair coat and blanches on diascopy. A large affected area is called angioedema. Examples include insect stings, positive intradermal skin test reactions and allergic reactions.

Secondary lesions

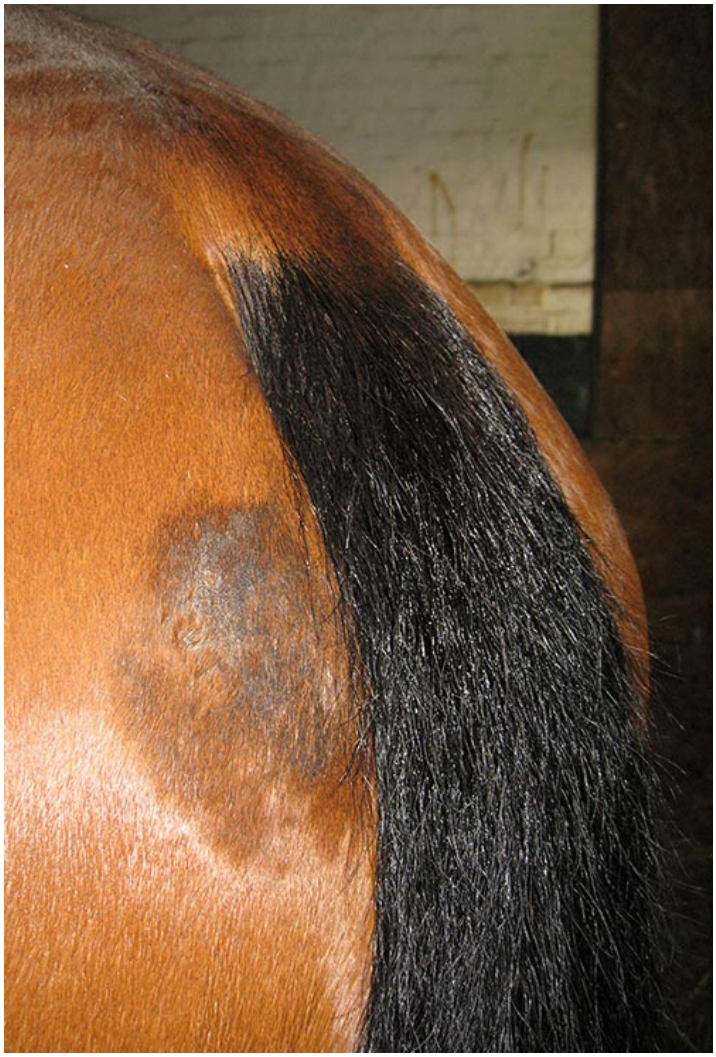

Figure 4. Rump rubbing due to sweet itch.

Evolving from primary lesions or artefacts induced by the patient, or external factors, such as trauma and medications.

Epidermal collarette

Scale arranged in a circular rim – the remnants of a vesicle/pustule/papule/bulla, scar, excoriation.

Scar

Area of fibrous tissue replacing traumatised/damaged dermis or SC tissue.

Excoriation

Erosions/ulcers from scratching/biting/rubbing – usually due to pruritus with secondary infection.

Erosion

Shallow epidermal defect not penetrating the basal laminar zone due to epidermal diseases/self-inflicted trauma. Heals without scarring.

Fissure

Single/multiple lines of cleavage into the epidermis or dermis caused by disease/injury – thickened and inelastic skin exposed to swelling from trauma/injury – seen on the ears, nose and footpads.

Lichenification

Skin thickening/hardening with exaggeration of superficial skin markings – often the result of friction and may be hyperpigmented and have infection.

Callus

Thickened, rough, hyperkeratotic, alopecic, lichenified plaque, commonly over bony prominences due to pressure and chronic low-grade friction – for example, burns or deep pyoderma (normally alopecic, atrophic and depigmented).

Primary or secondary lesions

Table 4. Medical approaches to managing sweet itch		
Approach	**Examples**	**Administration**
Antihistamines	Hydroxyzine	1mg/kg to 2mg/kg tid works well for urticaria
	Chlorpheniramine	Six tablets once a day works in some cases
Corticosteroids (care – laminitis)	Injectable	
	Oral	Prednisolone 0.25mg/kg to 0.5mg/kg
	Topical – spray	
	Creams/gel	
Antibiotics	Potentiated sulphonamides	
Feed supplements	Nicotinamide	Treatment should be started early in the year before symptoms develop to be effective
Allergen-specific immunotherapy (desensitisation)		Several studies have been carried out to try to desensitise horses and ponies with sweet itch using allergy vaccine injections and oral capsules
Immune modulation		A weekly course of oral capsules based on a suspension of heat-killed bacilli. Can be ordered directly by the public online via www.itchyhorse.co.uk

Table 4. Medical approaches to managing sweet itch.

Some lesions may be primary or secondary.

Alopecia

Partial or complete hair loss.

Primary causes: endocrine/follicle dysplasia

Secondary causes: trauma/inflammation

Scale

Accumulation of loose fragments of the horny layer (stratum corneum), dry or greasy.

Primary lesion with: colour dilute alopecia, follicular dysplasia, idiopathic seborrhoea and ichthyosis

Secondary lesion with: chronic inflammation

Crust

Dried exudate/serum/pus/blood cells/scales/medication adhered to skin surface.

Primary lesion with: idiopathic seborrhoea and zinc-responsive dermatosis

Secondary lesion with: pyoderma, myiasis and pruritus

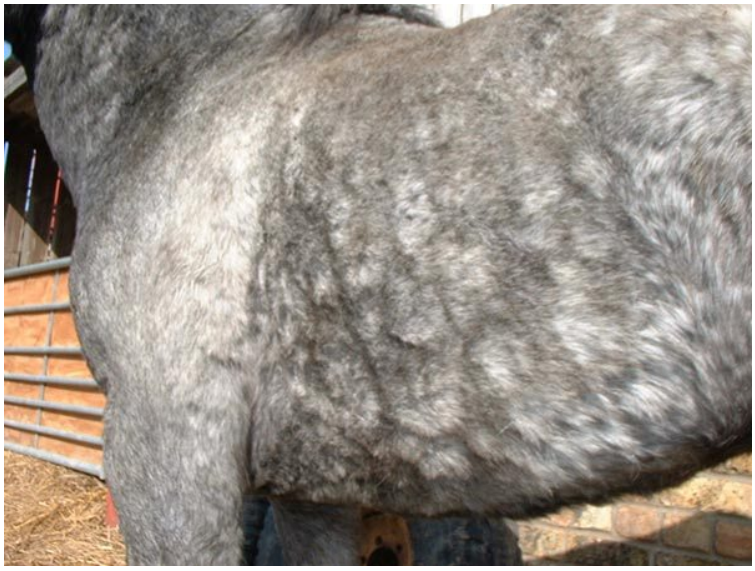

Figure 5. Multiple raised lumps (wheals) on the body after the administration of a wormer.

Follicular casts

Accumulation of keratin and follicular material adhering to the hair shaft.

Primary lesion with: vitamin A-responsive dermatosis, idiopathic seborrhoea and sebaceous adenitis

Secondary lesion with: demodicosis and dermatophytosis

Comedo

Dilated hair follicle filled with cornified cells and sebaceous material.

Primary lesion with: vitamin A-responsive dermatosis, Cushing's disease, sex hormone dermatoses and idiopathic seborrhoea

Secondary lesion with: seborrhoea, occlusion with medication, hair follicle diseases – demodex/dermatophytes

Pigmentary abnormalities

- Skin discolouration:
  - black = melanin (if throughout the epidermis, lentigo)
  - red/purple = haemorrhage (bruise)
  - yellow = icterus – bile pigmentation

- Leukoderma – white skin, leucotrichia – white hair
- Hypopigmentation/hypomelanosis

Primary lesion with: vitiligo-like disease

Secondary lesion with: post-inflammatory change

Categorising skin lesions this way allows the list of differential diagnoses to be narrowed down and a specific diagnosis reached more quickly.

Primary lesions are the first result of hypersensitivity reactions, such as nodules, papules, pustules and wheals. These are then altered by self trauma, with the skin becoming further damaged by excoriation, producing secondary lesions such as scales, crusts, alopecia, lichenification and hyperpigmentation.

Case studies

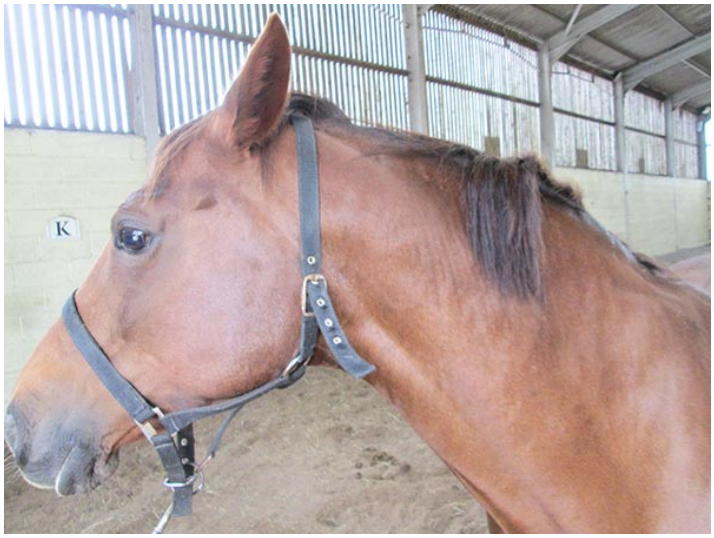

Figure 6. Megan, a 12-year-old Welsh cross mare that had been scratching for six months.

The horse shown in **Figure 2** developed a contact allergy. It was normally kept on barley straw, but developed wheals and urticaria a short time after it came into contact with wheat straw.

When it was apparent the problem had arisen after the change to a different bedding, the new bedding was removed, a cortisone injection was given to settle the lesions and the problem resolved.

Insect stings can also cause hives or urticaria and large swellings can appear all over the body. In

the case shown in **Figure 3**, the head had swollen up and the nose band of the head collar had left an impression where it had become tight.

This horse was quite distressed by the lesions, but once given a cortisone injection, the problem resolved and the horse became a lot more comfortable.

Some insects, such as midges (*Culicoides*) and black flies, need to feed on animal blood and, while feeding, will leave a small amount of the anticoagulant saliva in the skin, which is normally absorbed by the body.

However, some individual animals have an immune system that causes an allergic reaction to the insect saliva, causing the skin to become very itchy. The animal then rubs and scratches itself, making large sore patches. An example of this in horses is sweet itch, where horses allergic to the insect bites rub themselves raw on the tail and mane, and under the belly (**Figure 4**).

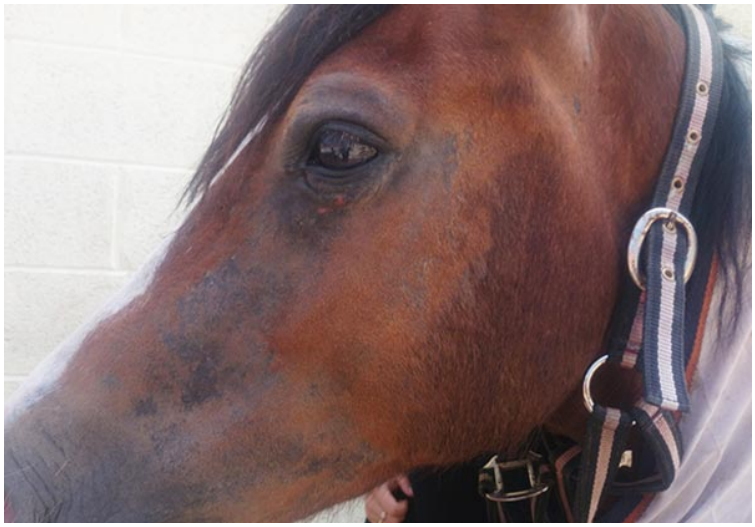

Figure 7. Eight-year-old Welsh section D pony mare Cassie started developing skin lesions.

Sweet itch has a hereditary component and is seen particularly in certain breeds, such as Shires, and Icelandic and Welsh ponies.

The midges are particularly prevalent near areas of free-standing water and when low winds are apparent, such as in the early morning or late evening in summer and autumn. Management approaches to treating sweet itch are:

- Full-body blanket
- Keeping animals stabled from 4pm to 10am
  - Only keep affected individuals outdoors when midges aren’t about. Midges are crepuscular (active at dawn and dusk).

- Fitting a fly screen to the stable door
- Fitting a fan in the stable
  - Midges do not like air movement.

- Insect repellents
- Relocating the animals
  - Keeping away from standing water and trees.

- Applying oily topical skin preparations
  - Midges do not like biting through oil. Avon Skin So Soft has been reported to help.

Medical approaches, meanwhile, are summarised in **Table 4**.

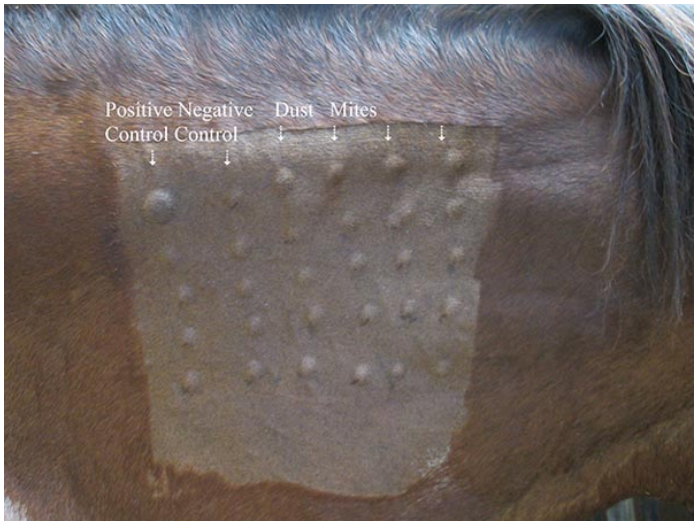

Figure 8. Megan’s intradermal skin test showing positive reactions to dust mites.

Some horses can be allergic to certain drugs. When given medications that normally don’t affect normal individuals, they react in an adverse way, potentially causing some serious results.

The case shown in **Figure 5** developed large areas of irregular bumps in the skin following the administration of a routine wormer. While the reaction went down after a while, the owner was careful to avoid any similar wormers again.

The predisposition to allergic symptoms following repeated exposure to inhalant allergens is called atopic dermatitis, with breeds such as Arabians and Thoroughbreds appearing to be predisposed to the condition (White, 2015).

Common allergens include pollens, moulds and dust mites, with the reaction causing intense itching.

The horse often then bites at itself and rubs on fences and stable walls, causing self-inflicted damage, hair loss and ulcerated skin lesions.

The disease can be investigated through allergy testing, where extracts of common environmental agents are injected into the skin and the reaction is measured (Littlewood, 1997).

The case study seen in **Figures 6 to 9** shows how two completely different allergic diseases can present with the same symptoms, but require different treatment approaches to resolve them. Megan and Cassie were two horses on the same yard at the same time and both were very itchy, rubbing themselves on fences and stable walls, which resulted in the horses having areas of alopecia and excoriation.

No evidence of ectoparasites, such as lice or mites, was found on skin scrapes, while fungal culture was negative for ringworm on both horses. Having ruled out infectious causes of their skin problems, further investigation with intradermal skin testing was carried out, where measured doses of extracts of common environmental agents were injected into the skin and their reaction compared to a positive and negative control.

Megan showed strong reactions to dust mites, indicating she had atopic dermatitis, while Cassie showed a strong allergic reaction to the *Culicoides* insect bites, but nothing else, indicating she had sweet itch.

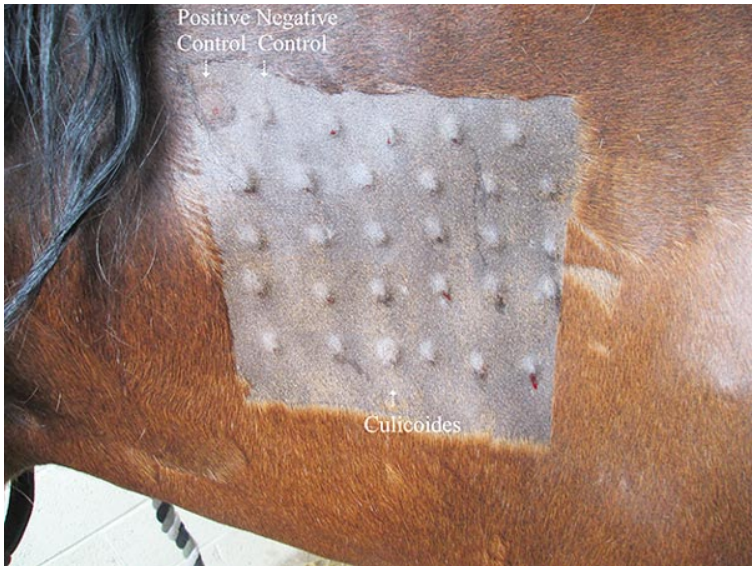

Figure 9. Cassie’s intradermal skin test showing a positive reaction to *Culicoides*.

The treatment for atopic dermatitis with Megan involved administering a short-acting steroid and antibiotics, but because minimising the exposure to dust mites wasn’t practical, she was desensitised with an allergy vaccine.

This involves administering small doses of the agents the individual is allergic to, gradually building up to larger and larger doses until the individual no longer develops an allergic reaction to them.

This treatment has been shown to be safe and effective, reducing the clinical signs of atopy in 84% of patients (Stepnik et al, 2012).

The treatment for sweet itch with Cassie involved following the management approaches in **Table 4** to minimise the exposure to the biting *Culicoides* midges, and the medical approach of giving a short-term steroid injection and course of antibiotics to treat the secondary skin infection (pyoderma).

Reducing the exposure of an animal to the allergens by regular shampooing with a mild soothing shampoo can also be beneficial, which – alongside corticosteroids in the short term – will suppress the sensitivity of the skin to the allergenic agents. However, long-term use of steroids can be harmful, with side effects including laminitis and liver damage.

A variety of allergic reactions that horses can develop exist, producing a range of clinical signs. Painstaking investigation to get to the bottom of the cause and produce an effective treatment plan is essential to managing their problems and allowing horses to continue a comfortable life.

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