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FOOD ALLERGY IN THE HORSE: A DERMATOLOGIST'S VIEW

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Definition

Food allergy is an uncommon and poorly understood disease in the horse. Symptoms can be gastrointestinal, dermatologic, or both. The terminology itself is confusing. The term food allergy implies an immunologic reaction to an ingested substance. Not all food allergies are truly allergic in nature. A better term for the condition would be "adverse reactions to food." This designation includes immunologic and nonimmunologic reactions to food substances. Nonimmunologic food sensitivities include metabolic, pharmacologic, and idiosyncratic reactions. An example of this would be pruritus resulting from the histamine content of a food (e.g., mackerel or tuna), instead of from an allergic reaction to the food itself. Fortunately, clinical symptoms and treatment of immunologic and nonimmunologic food reactions are identical. Therefore, for the sake of simplicity, most clinicians use the term food allergy to indicate adverse reactions to food.

Pathogenesis

Very little is known about the pathogenesis of food allergy in the horse. Most of the research into food allergy has concentrated on human food hypersensitivities, particularly in children. True food allergies in humans have been classified as type I hypersensitivity reactions for the most part, but type III and IV hypersensitivity reactions are also suspected. Type I reactions (immediate type hypersensitivity) encompass all IgE-mediated reactions such as pruritus, erythema, and urticaria. Type III (arthus) hypersensitivities typically cause vasculitic lesions. Type IV reactions (delayed type hypersensitivity) usually cause a papular eruption. All three types of clinical reactions have been observed in the food-allergic horse, but detailed information about the pathogenesis of each type of reaction is lacking. Unfortunately, at this time much of the information known about food allergy in the horse is case-based and anecdotal.

The first stage of any hypersensitivity reaction is the sensitization phase. During sensitization, the antigen is repeatedly presented to T lymphocytes and an abnormal hypersensitivity response occurs instead of tolerance. For a food allergy to occur, an allergen must breach the intestinal mucosal barrier in order to be exposed to the

immune system. Normally, the intestinal tract has various mechanisms to prevent the absorption of potentially allergenic substances. Intestinal enzymes break down large molecules into smaller, less antigenic ones. The mucous coating and tight junctions of the intestinal epithelium do not allow larger, more antigenic macromolecules to pass the intestinal barrier and reach the immune system. Secretory IgA binds to smaller antigenic molecules, thereby preventing their penetration of the mucosa. Intestinal peristalsis also helps by moving large molecules through the intestinal tract at a relatively rapid rate.

Hypersensitivity can result when there is a break in intestinal barrier function and an abnormal immune response to a presented antigen. This can occur in young animals whose intestinal mucosal barrier is not fully developed, in old animals whose intestinal mucosal barrier has begun to degenerate, and in animals with gastrointestinal illnesses that have damaged the intestinal barrier. In these cases, macromolecules (>12 kilodaltons) are able to pass through the mucosal barrier and reach the immune system. Under normal circumstances, especially in the young animal, a CD8 positive T cell response occurs and results in tolerance/anergy. However, if Th2 lymphocytes are activated, a type I hypersensitivity reaction can develop. The reason this abnormal immunologic response occurs in some individuals is still unknown. It may be genetically determined, or may occur secondary to another event such as a heavy parasite load. Parasite antigens cause activation of many T helper cells, some of which may inadvertently react to food antigens instead of parasite antigens. Another possibility is the formation of a hapten-antigen complex. This can occur when a small, theoretically nonantigenic molecule is easily absorbed through the mucosal barrier, then combines with a hapten to form a complex that is now large enough to elicit an immunologic response.

In the diet, water-soluble glycoproteins are thought to act as the base of many of these antigens. These glycoproteins can be found in fresh or prepared foods, supplements, or other additives. Unfortunately, what is not known is which glycoprotein(s) in a particular food item is important, or what part of each particular glycoprotein is antigenic.

Incidence

The true incidence of adverse food reactions in the horse is not known. It is assumed to be uncommon; however, it can be difficult to rule out, so many food-allergic horses may not be properly diagnosed. It is also possible that the condition is recognized and corrected by an owner prior to seeking veterinary attention, so we have no knowledge of these cases. Diet items reported to cause adverse food reactions in horses include alfalfa, barley, beet pulp, bran, buckwheat, chicory, clover, malt, oats, potatoes, St. John's wort, wheat, feed additives, and feed supplements.

History

Obtaining a good history, while sometimes difficult, is crucial for the diagnosis and management of food allergy. The person who is responsible for the day-to-day care, feeding, and grooming of the patient must be interviewed. This is not necessarily the owner of the horse. Important historical information to ascertain includes the patient's age at onset, time of year of onset of condition, seasonality, environmental conditions, and changes in environment and their effect on symptoms. Gather as much information as possible about the horse's past and current diet. Commercial feeds, hay types, pasture, treats, and supplements/additives must be recorded. It is also important to note if the horse's diet is consistent throughout the year. Other information that should be obtained includes previous treatments and response (if any), current treatment and response, insect control measures, intestinal parasite control measures, and any other current/past illnesses.

Food allergies can start at any age (1 year to 10 years). Symptoms of food allergy are usually nonseasonal and do not vary with changes in environment (provided the diet is not changed).

Clinical Signs

Clinical signs of food allergy are extremely variable both in the horse and other species. Food allergy is usually pruritic, but nonpruritic urticaria is also reported. Other dermatologic signs seen with adverse food reactions include angioedema, papules, excoriations, erythema, crusts, alopecia, and vasculitic lesions. Gastrointestinal symptoms may also be present. Since the signs of food allergy are not unique, other dermatologic diseases must be eliminated from the differential diagnosis list before the diagnosis of food allergy is made.

Various mites (sarcoptic, psoroptic, chorioptic) can cause a typically nonseasonal pruritic dermatosis. Most of these parasites can be found on skin scraping. Trial therapy with ivermectin can also be helpful in ruling out parasitic infections. Dermatophytosis, bacterial folliculitis, and dermatophilosis can all cause a severe, nonseasonal pruritic dermatitis that can look identical to food allergy. Fungal culture, bacterial culture, skin surface cytology, and skin biopsy can be used to diagnose these infections.

Food allergy must also be differentiated from other allergic diseases such as insect bite allergy, contact allergy, and atopy. Contact allergic reactions almost always have a primary eruption (papules, pustules, erythematous macules, wheals, erythematous plaques). The eruption may be seasonal (if plant related) or nonseasonal (topical medications, shampoos, tack cleaners). The distribution of lesions can often give clues to the presence of a contact reaction. Insect bite allergy (*Culicoides* hypersensitivity, other biting flies) typically results in a severely pruritic, papular dermatitis that is most often seasonal. Atopy (inhalant allergic dermatitis) can cause mild to severe pruritus.

A primary eruption (papules, pustules, erythematous macules, erythematous plaques) is not always present, and pruritus varies from mild to severe. Atopy can be seasonal or nonseasonal, depending on geographic location.

Diagnosis

The only reliable way to confirm a diagnosis of food allergy is with an elimination diet. Both intradermal allergen testing and serum testing have been shown to be unreliable for identifying food allergies. These diagnostic tests have been investigated in other species, and were found to produce an unacceptable number of false positives and false negatives. Intradermal allergen testing is useful for identifying important allergens in atopic individuals. The diagnosis of atopy is made by history, clinical signs, and exclusion of other pruritic dermatoses. The diagnosis of atopy is not made by intradermal allergen testing. Some of the pollens on an intradermal testing panel are from grasses that are used for hay. A positive reaction on an intradermal test does not mean this grass is a food allergen for this individual. However, most hay will contain pollen from the grass, as well as a variety of other pollens, dust, etc. For this reason, a change in the type of hay being fed may be recommended. At the very least, the hay should be fed on the ground and lightly misted with water to minimize dust.

A good elimination diet consists of a single protein source and carbohydrate source to which the patient has had no previous exposure. Finding a novel protein and carbohydrate source can be particularly difficult in the equine patient. In addition, all supplements and dietary additives must be discontinued. Limiting the diet to fresh hay from a different grass (e.g., timothy to alfalfa) and offering a single-ingredient pelleted diet is often the best option available. Dietary manipulation can be difficult to impossible in performance horses.

The food trial should be continued for 8 to 12 weeks to see maximal improvement. Most food allergic individuals show improvement in clinical signs in 4 to 6 weeks. If clinical improvement is noted, the patient can be challenged with items from its previous diet. One new item should be introduced every week. This allows the offending diet items to be identified.

Management

The only effective treatment for food allergies is avoidance of the offending allergen. Antihistamines and glucocorticoid therapy can be tried, but are not particularly effective. The lack of readily available, prepared feeds that consist of a limited number of ingredients makes management of food allergic horses difficult. A variety of single-ingredient feeds designed for the various life stages and activities of horses would make diagnosing and managing equine food allergies much easier.

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