Zinc Responsive Dermatosis

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Zinc responsive dermatosis in dogs occurs due to an impaired ability to absorb zinc from the gut (type 1) or a relative or absolute deficiency of zinc in the diet (type 2). Naturally occurring zinc deficiency has not been reported in cats.

Type 1 disease is most frequently seen in Siberian Huskies, Samoyeds and Alaskan Malamutes (possibly due to autosomal recessive inheritance), although it has been recognized in other breeds. These animals appear unable to absorb adequate zinc, even when fed a nutritionally balanced diet.

Type 2 disease, due to an absolute dietary zinc deficiency, is rare in animals fed high-quality, commercially-prepared diets. More commonly, there is a relative deficiency due to interaction with other dietary components or an inability to utilize dietary zinc. Absorption of zinc from the gut is inhibited by iron, copper, and calcium. Phytate and inorganic phosphate bind zinc and hinder absorption in the intestine. This is most likely to be seen in rapidly growing animals, particularly giant breeds, fed inadequate diets or diets in which nutritional antagonism occurs, particularly due to high phytate content or over-supplementation with calcium.

There is no sex predisposition, although clinical lesions may be associated with or exacerbated by estrus, pregnancy, and lactation in intact females. Most cases of type 1 disease are seen between 1 and 3 years of age, although there is a wide age range of up to 11 years at first presentation. Type 2 is normally seen in young, growing dogs fed inappropriate diets, although it can be seen in older animals depending on the dietary history.

Cutaneous lesions include well-demarcated, symmetrical areas of scaling, crusting, lichenification and erythema predominantly around the mouth, eyes, pressure points and scrotum. Pruritus is variable, but it may be severe. Affected skin may fissure and ulcerate, which is often painful. Secondary pyoderma and yeast dermatitis may occur. The coat is generally dull and harsh and may exhibit multifocal hypopigmentation. Other clinical signs include lymphadenopathy (especially if there is fissuring, inflammation, and/or pyoderma), poor wound healing, anestrus, infertility, inappetence (possibly due to altered taste and/or smell), failure to thrive, and weight loss.

DIFFERENTIAL DIAGNOSES

- - Atopic dermatitis
- - Adverse reaction to food
- - Demodicosis
- - Superficial pyoderma
- - Malassezia dermatitis

• - Dermatophytosis
• - Pemphigus foliaceus
• - Leishmaniasis

The history (particularly the breed and diet) and clinical signs are highly suggestive. Cytology reveals numerous nucleated keratinocytes consistent with widespread parakeratosis, with or without yeast and/or bacteria and neutrophils. Histopathology will confirm acanthosis with diffuse parakeratosis. Parakeratosis, however, may be focal or minimal in some cases. Low zinc levels in plasma or hair are supportive, but there is a wide overlap with normal dogs and false-negative results due to zinc contamination from reagents and equipment are common. Plasma and hair zinc levels are generally the least useful information in the diagnosis of this disease. Final confirmation of the diagnosis relies on response to treatment.

The prognosis is generally good. Therapy involves correction of any dietary factors and supplementation with zinc methionine (4mg/kg/day PO). Higher doses may be necessary in some dogs. Zinc sulfate can cause vomiting and diarrhea, so zinc gluconate or methionine are generally preferred. Higher doses produce a better response, especially initially, but these doses may be less well tolerated. Anecdotal evidence indicates that treatment with essential fatty acids (EFAs) and oral glucocorticoids may dramatically enhance response to therapy. It is unclear whether this is associated with enhanced uptake or utilization of zinc or reduction of cutaneous inflammation.

Secondary yeast or bacterial dermatitis is common in dogs with zinc responsive dermatosis. Surface cytology is required to detect these secondary infections.

The most common cause of treatment failure is improper dosing of zinc. Zinc supplementation is required for life in type 1 disease, whereas it should be possible to maintain affected animals on a nutritionally balanced diet once the clinical signs have resolved in type 2 disease.

KEY POINTS
• - May occur in dogs on a commercially prepared diet given calcium and/or cereal supplements.
• - This condition is variably pruritic.