

Zinc Responsive Dermatitis

AETIOLOGY AND PATHOGENESIS

Zinc responsive dermatosis in dogs occurs due to an impaired ability to absorb zinc from the gut. The disease is most frequently seen in Siberian Huskies and Alaskan Malamutes (possibly due to autosomal recessive inheritance), although it has been recognised in other breeds. These animals appear unable to absorb adequate zinc, even when fed a nutritionally-balanced diet.

CLINICAL FEATURES

There is no sex predisposition, although clinical lesions may be associated with or exacerbated by oestrus, pregnancy and lactation in intact females. Most cases are seen between 1-3 years of age, although there is a wide age range up to 11 years at first presentation.

Cutaneous lesions include well-demarcated, symmetrical areas of scaling, crusting, and erythema predominantly around the mouth, the eyes, and pressure points and some times the lower legs. Pruritus is variable but may be severe. Affected skin may fissure and ulcerate, which is often painful. Secondary bacterial infection is common. 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 bacterial infection), poor wound healing, anoestrus, infertility, inappetence (possibly due to altered taste and/or smell), failure to thrive and weight loss.

DIFFERENTIAL DIAGNOSES

- Superficial bacterial infection
- Yeast dermatitis
- Demodectic mange
- Ringworm
- Pemphigus foliaceus
- Superficial necrolytic dermatitis (migratory necrolytic erythema or hepatocutaneous syndrome)

DIAGNOSTIC TESTS

The history (particularly the breed) and clinical signs are highly suggestive. Biopsy which features extensive parakeratosis will confirm the diagnosis. 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. Final confirmation of the diagnosis relies on response to treatment.

MANAGEMENT

The prognosis is generally good. Therapy involves correction of any dietary factors and zinc supplementation. Higher doses may be necessary in some dogs. Zinc sulphate can cause vomiting and diarrhoea, so zinc gluconate or methionine are often preferred. There is a better response to higher doses, especially initially, but these may be less well tolerated. The most common cause of treatment failure is to base the dose on the zinc compound, and not the elemental zinc content. There is anecdotal evidence that treatment with essential fatty acids and prednisolone may speed the clinical response, although it is unclear whether this is associated with enhanced uptake or utilisation of zinc or amelioration of cutaneous inflammation. Antibiotics may be necessary to control secondary infections. Zinc supplementation may be required for life. Topical steroid preparations may aid in the control of lesions in some cases.

KEY POINTS

- Rare condition which can only be controlled and not cured
- If there is poor response the dose of zinc may have to be increased.