

Metabolic Epidermal Necrosis

AETIOLOGY AND PATHOGENESIS

Metabolic epidermal necrosis is an uncommon skin disorder variously known as diabetic dermatopathy, hepatocutaneous syndrome, necrolytic migratory erythema and superficial necrolytic dermatitis. It is associated with metabolic diseases such as liver diseases, diabetes mellitus and pancreatic tumours. It is uncommon in dogs and very rare in cats.

Abnormalities of the liver characterised by moderate to severe vacuolation of hepatocytes, parenchymal collapse, and nodular regeneration are present in the majority of cases. Most animals with liver disease, however, do not have epidermal metabolic necrosis, which suggests that the pathogenesis involves a specific underlying metabolic dysfunction rather than primary liver disease. Hyperglycemia and diabetes mellitus is noted in many dogs, but this tends to occur after hepatic and cutaneous disease and is not therefore thought to be involved in the pathogenesis. Most dogs have markedly decreased plasma amino acid levels, which may lead to epidermal protein depletion and death of skin cells.

CLINICAL FEATURES

Epidermal metabolic necrosis is a disease of older dogs, and cutaneous changes generally precede systemic illness. No sex predisposition has been noted, although the condition may be more frequent in West Highland White Terriers and Shetland Sheepdogs. Some dogs may have a history of weight loss. Hyperkeratosis, scaling, crusting, and cracking of the digital pads are the most consistent clinical findings, and may result



in lameness. The scales are typically large, thick and tightly adherent. Redness of the skin, scaling, erosion, ulceration and crusting occur on the muzzle, junctions of skin and lips, ears, pressure points (elbows, hocks, hips, and stifles), genitalia, abdomen and axillae. Ulceration of the oral cavity is

seen in some cases. Dullness, inappetance and increased thirst may occur in the later stages with overt hepatic failure and/or diabetes mellitus. Secondary bacterial and yeast infections are common.

DIFFERENTIAL DIAGNOSES

1. Pemphigus foliaceus
2. Cutaneous or systemic lupus erythematosus
3. Zinc responsive dermatosis
4. Superficial bacterial or fungal infections
5. Lymphoma of the skin (a type of cancer of the skin)
6. Primary keratinisation defect
7. Demodectic mange

DIAGNOSTIC TESTS

The history and clinical signs are highly suggestive. Histopathology of skin biopsies is often diagnostic. Several biopsies may be necessary, as findings are variable and inconsistent. Blood glucose levels are generally elevated, and liver function tests are abnormal. These parameters may, however, be normal. Plasma amino acids may be low, and plasma glucagon may be elevated if a glucagonoma is present. Affected livers have a typically 'honeycomb'-like appearance on ultrasonography. Further investigation including ultrasound guided biopsy may be necessary to confirm liver pathology consistent with hepatocutaneous syndrome. Other tests may be required to detect a glucagonoma or diabetes mellitus.

MANAGEMENT

Metabolic epidermal necrosis, due to causes other than glucagonoma, is associated with serious internal disease and the prognosis is poor with most dogs dying or being euthanased within five months of the development of cutaneous lesions. Despite this, aggressive therapy may result in prolonged survival times for a year or more in some dogs.

Critical care or liver specific prescription diets with high quality proteins should be fed as the main diet if the dog will accept a change of food. High quality protein supplements such as 3-6 egg yolks, powdered casein or proprietary amino acid combinations can be added to the diet. Intravenous amino acids have been very successful in some dogs. Dogs being treated with amino acids should be monitored for signs of hepatic encephalopathy and plasma ammonia should be monitored every 2-4 hours if possible. Other therapies for hepatic disease that may be of benefit include s-adenosylmethionine and ursodeoxycholic acid, although the evidence is largely anecdotal.

If a glucagonoma is diagnosed, surgical removal is the treatment of choice, although the surgery can be difficult and is associated with significant post-operative death.

Hair should be clipped from moist lesions to avoid matting and crusting. Gentle bathing with anti-scaling and/or antibacterial shampoos will facilitate resolution of secondary infections and open lesions.

KEY POINT

- History and clinical signs are usually enough to suggest the diagnosis, but skin biopsies, liver function tests, liver biopsy and ultrasound may be necessary. The prognosis is guarded to poor but prolonged survival times may be achieved with aggressive therapy in some cases.