Ceramides & Canine Atopy

A study of human atopic dermatitis (AD) showed obvious reduction of protein-bound ceramides in AD skin compared with normal skin. The canine stratum corneum (SC) has a pattern of free and protein-bound ceramides like that in humans. The SC of atopic dogs shows decreased free lipids and a highly significant reduction of protein-bound fatty acids and ceramides compared to normal canine SC. Large amounts of glucosylceramides are present, although they are nearly absent in normal canine SC.

This study analyzed the changes that occurred in SC lipids of dogs after 3 weeks of topical treatment with an emulsion (skin lipid complex [SLC]) containing ceramides, free fatty acids (FFAs), and cholesterol. Nonlesional SC was collected from control and treated areas of both atopic and healthy dogs. Free and protein-bound lipids were purified, analyzed, and assayed. Levels of cholesterol and FFAs were lower in atopic dogs, although differences were not significant. Total ceramides were significantly reduced in untreated atopic SC, and high levels of glucosylceramides were found, in contrast to their near absence in normal SC. Free ceramide content of atopic SC was significantly increased after treatment, near that of normal canine SC; glucosylceramides were barely detectable. Topical treatment with SLC resulted in significant improvement of lipid biosynthesis of keratinocytes in atopic dogs, potentially enabling the formation of a tighter epidermal barrier.

Commentary

The clinical features of human and canine AD are similar and appear to result from an impaired skin barrier, resulting in increased transepidermal water loss. In this study, twice-weekly application of a sphingolipid-containing emulsion resulted in significant increases in free ceramides and normalization of glucosylceramides within the SC of atopic dogs. There were also increases in some protein-bound ceramides within treated SC, suggesting treatment with this emulsion may stimulate biosynthesis of some ceramides rather than simply replenishing deficient quantities. Further studies are needed to show whether repair of deficient skin barrier function in atopic dogs results in reduced clinical disease.—*Lisa Akucewich, DVM, DACVD*

Source

The lipid alterations in the stratum corneum of dogs with atopic dermatitis are alleviated by topical application of a sphingolipid-containing emulsion. Popa I, Remoue N, Osta B, et al. *CLIN EXP DERMATOL* doi: 10.1111/j.1365-2230.2011.04313.x.

FIP Update: Hope for the Future

Feline coronavirus (FCoV) consists of two biotypes: feline enteric coronavirus (FECV), causing mild systemic signs, and feline infectious peritonitis virus (FIPV), causing FIP. Two theories on FIP development exist. The *mutation theory* maintains that FIPV mutates from FECV in an infected cat and no horizontal transmission occurs. The *circulating virulent-avirulent FCoV hypothesis* claims that FECV and FIPV strains circulate in the environment, and FIP only results from infection with the FIPV strain. New developments indicate that the vasculitis occurring with clinical disease is driven by monocytes, and that endothelial cells can assume a primary role. In the wet (effusive) form of FIP, vasculitis is enhanced by antigen-antibody immune complex formation, resulting in antibody-dependent enhancement of monocyte infection and effusive disease. In the dry (proliferative) FIP form, a mixed Type I/Type II immune response results in granuloma and plaque formation on serosal surfaces of organs.

Concentrations of the acute phase protein (ie, α-1-acid glycoprotein) >3000 µg/mL are supportive of, but not definitive for, diagnosis. High globulin levels and a decreased albumin:globulin ratio are found most consistently. Fluid analysis can be useful. Histopathology remains the gold standard for diagnosis. Treatment is supportive and symptomatic, including immunosuppressive and antiinflammatory drugs. Although criteria for remission exist, FIP is usually progressive and fatal, and humane euthanasia may be considered when refractory disease is present.

Commentary

Despite much research about the natural biology of the causative virus and the disease, FIP remains a poorly understood and potentially frustrating disease. This welcome update reviewed how and why FIP develops in certain kittens but not in others, as well as the reliability of testing and new treatment approaches, such as polyprenyl immunostimulant. Further investigation for FIP treatment includes small interfering RNA molecules and drugs inhibiting the ability of the virus to evade immune response. Future combinations of therapeutic approaches may make FIP, particularly noneffusive FIP, a chronically manageable disease.—*Susan Little, DVM, DABVP (Feline)*

Source

Update on feline infectious peritonitis. German A. *IN PRACT* 34:282-291, 2012.

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