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WHEN THE SKIN SPEAKS: UNDERSTANDING ATOPIC DERMATITIS

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Introduction

Atopic dermatitis (AD) is one of the most common dermatological diseases in small animal clinics, being the main cause of chronic pruritus in dogs and cats. Its prevalence in the general canine population is around 10–15%, and more than 50% of atopic patients develop otitis externa. AD is a complex syndrome of multifactorial origin, with a genetic predisposition and close interaction between the skin barrier, the microbiome and the immune response.

Skin barrier and microbiome

Healthy skin acts as a physical and functional barrier against pathogens, allergens and UV radiation, as well as maintaining water and lipid homeostasis. In AD, alterations in the skin barrier are observed: increased transepidermal water loss (TEWL), decreased ceramide content and mutations in structural proteins such as filaggrin. These alterations facilitate the penetration of allergens and microorganisms, triggering inflammation. The skin microbiome also plays a central role, with a decrease in bacterial diversity and an increase in colonisation by pathogens such as *Staphylococcus pseudintermedius and Malassezia pachydermatis.* These dysbiosis contribute to the maintenance of the inflammatory process.

Pruritus and pathophysiology

Pruritus is the cardinal clinical sign of atopic dermatitis and results from a complex neuroimmunological interaction. Allergens cross the altered skin barrier, activating keratinocytes, mast cells and T lymphocytes that release pruritogenic cytokines, mainly IL-31, together with IL-4 and IL-13, mediators of the Th2 response. These cytokines activate sensory nerve fibres that release neuropeptides such as substance P, maintaining the vicious cycle of inflammation–itching–scratching. The intracellular signalling of this cascade depends to a large xtent on the JAK/STAT pathway, where JAK1 plays a key role in the transmission of itch and allergic inflammation signals.

Diagnosis

The diagnosis of AD is based on a combination of medical history, clinical examination and exclusion of other pruritic diseases. The Favrot criteria are useful as a diagnostic tool in dogs and cats, with sensitivities and specificities greater than 80%. Laboratory diagnosis involves elimination and challenge diets to rule out adverse reactions to food, along with allergy tests such as intradermal testing or specific serum IgE detection, aimed at identifying environmental allergens and establishing specific immunotherapy, the only treatment capable of hyposensitising against the cause.

Treatment

The management of atopic dermatitis should be multimodal and adapted to the inflammatory phenotype of each patient. It includes measures aimed at: restoring skin barrier function (ceramides, essential fatty acids, specific shampoos), controlling microbial dysbiosis (oral and topical antiseptics and pre/pro/postbiotics), reducing itching and inflammation (glucocorticoids, cyclosporine, JAK inhibitors), and hyposensitising against allergens through specific immunotherapy. The therapeutic goal should focus on preserving long-term skin health, with skin-friendly treatments that reduce side effects and improve the quality of life of the patient and their guardian.

Conclusions

Canine and feline atopic dermatitis is a complex, multifactorial syndrome that requires a comprehensive diagnostic and therapeutic approach. Understanding alterations in the skin barrier, the role of the microbiome, and the mechanisms of pruritus has led to advances in more specific and effective therapies. Clinical success depends on a multimodal, individualised and sustained approach aimed at reducing pruritus, controlling inflammation and improving quality of life.