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Histopathological characterization of canine atopic dermatitis: Observations from six clinical cases

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Abstract

Canine atopic dermatitis (CAD) is a chronic, pruritic, immune-mediated skin disorder characterized by epidermal barrier dysfunction, Th2-skewed cytokine responses, and hypersensitivity to environmental allergens. This study examined histopathological features in skin biopsies from six dogs clinically diagnosed with CAD at the Department of Veterinary Medicine, Veterinary College, Bengaluru. Microscopic evaluation revealed consistent patterns of spongiosis, apoptotic keratinocytes, mast cell activation, and peri-adnexal lymphocytic infiltration, reflecting the immunopathological mechanisms underlying CAD. In select cases, bullae formation and epidermal separation were observed, mimicking autoimmune blistering conditions and indicating severe barrier compromise. Dermal infiltrates included eosinophils, lymphocytes, plasma cells, and mast cells, supporting chronic antigenic stimulation and hypersensitivity. Additional findings such as epidermal hyperplasia and lymphocytic hidradenitis highlighted chronicity and follicular involvement. No evidence of neoplasia was detected. These results reinforce the diagnostic value of histopathology in CAD, complementing clinical scoring systems and immunological profiling. Understanding tissue-level changes enhances diagnostic accuracy and informs therapeutic strategies, particularly as cytokine-targeted biologics become integral to CAD management.

Keywords: CAD, histopathology, allergy, Favrot's criteria

Introduction

Canine atopic dermatitis (CAD) is a genetically predisposed, chronic, pruritic, inflammatory skin disease associated with IgE-mediated hypersensitivity to environmental allergens such as house dust mites, pollens, and molds (DeBoer & Hillier, 2001; Halliwell *et al.*, 2006) ^[1, 5]. It is one of the most prevalent dermatological conditions in dogs, with a complex pathogenesis involving epidermal barrier dysfunction, immune dysregulation, and microbial colonization (Leung *et al.*, 2004; Marsella, 2021) ^[7, 8]. The disease is characterized by symmetrical distribution of lesions, recurrent pruritus, and responsiveness to glucocorticoids, as outlined in Favrot's criteria (Nuttall *et al.*, 2013) ^[10]. Immunologically, CAD is driven by a Th2-skewed response, with elevated levels of cytokines such as IL-4, IL-13, and notably IL-31, which plays a central role in pruritus and inflammation (Dillon *et al.*, 2004; Neis *et al.*, 2006) ^[3, 9]. IL-31 acts through IL-31RA and OSMR receptors on keratinocytes and sensory neurons, contributing to itch and barrier disruption (Santoro *et al.*, 2020) ^[12]. Histopathological evaluation provides critical insights into the cellular and structural changes underlying CAD, including spongiosis, eosinophilic infiltration, mast cell activation, and peri-adnexal lymphocytic inflammation (Scott *et al.*, 1995; Kim *et al.*, 2015) ^[13, 6]. While clinical scoring systems such as CADESI-04 and pruritus visual analogue scale (pVAS) are essential for monitoring disease progression, histopathology remains a valuable adjunct for confirming diagnosis, ruling out mimicking conditions, and characterizing chronicity (DeBoer & Marsella, 2001; Eisenschenk *et al.*, 2024) ^[2, 4]. This study consolidates histopathological findings from six dogs clinically diagnosed with CAD, highlighting the diversity of microscopic lesions and their relevance to disease pathophysiology.

Materials and Methods

This study was conducted at the Department of Veterinary Medicine, Veterinary College, Bengaluru, involving six client-owned dogs clinically diagnosed with Canine Atopic Dermatitis (CAD). Diagnosis was confirmed using Favrot's criteria, which include early age of onset, indoor lifestyle, glucocorticoid-responsive pruritus, symmetrical lesion distribution, and exclusion of other pruritic dermatoses (Halliwell *et al.*, 2006; Nuttall *et al.*, 2013) [5, 10]. All dogs exhibited characteristic signs of CAD, including erythema, alopecia, excoriation, and lichenification, primarily affecting the face, ventral neck, abdomen, and limbs.

Skin biopsies were collected from active lesion sites using 4 mm punch instruments under aseptic conditions and local anesthesia. Samples were fixed in 10% neutral buffered formalin and submitted to VetLesions™ Veterinary Diagnostic Laboratory, Bengaluru, for histopathological evaluation. Each specimen was processed routinely and stained with hematoxylin and eosin. Microscopic analysis focused on epidermal architecture, dermal inflammatory infiltrates, adnexal structures, and cellular morphology. Specific attention was given to identifying features such as spongiosis, bullae formation, mast cell activity, and peri-adnexal lymphocytic infiltration. All histopathological evaluations were performed by board-certified veterinary pathologists (MVSc, PhD), and clinical correlation was advised in each case. The findings were interpreted in the context of CAD pathophysiology, with emphasis on immune-mediated changes and chronicity markers. No evidence of neoplasia was observed in any specimen.

Results

Histopathological evaluation of skin biopsies from six dogs clinically diagnosed with canine atopic dermatitis revealed a consistent pattern of inflammatory and structural changes indicative of chronic allergic skin disease. The epidermis frequently exhibited spongiosis, apoptotic squamous epithelium, and in some cases, bullae formation at the dermal-epidermal junction, suggesting barrier disruption and intraepidermal edema. These features are consistent with cytokine-mediated epidermal damage, particularly involving IL-31 and IL-4 pathways (Dillon *et al.*, 2004; Santoro *et al.*, 2020) [3, 12]. The dermis showed diffuse infiltration of eosinophils, lymphocytes, plasma cells, and mast cells, with occasional neutrophilic presence. Mast cells displayed anisocytosis, anisokaryosis, and nuclear pleomorphism, with indistinct cytoplasmic boundaries and metachromatic granules, reflecting hypersensitivity-driven activation. In several specimens, peri-follicular lymphocytic infiltration and lymphocytic hidradenitis were observed, particularly around sweat glands and hair follicles, indicating chronic antigenic stimulation and follicular involvement (Kim *et al.*, 2015; Neis *et al.*, 2006) [6, 9]. One subset of samples showed benign hyperplastic proliferation of the epidermis with acanthosis, while another revealed complete separation of the epidermis from the dermis, mimicking autoimmune blistering conditions such as bullous pemphigoid. These findings, though variable, were consistently aligned with the clinical diagnosis of CAD and its immunopathological basis. No evidence of malignancy was found in any specimen.

Collectively, these histopathological features—spanning epidermal disruption, eosinophilic and lymphocytic infiltration, mast cell activation, and adnexal inflammation—support the diagnosis of canine atopic dermatitis and reflect its multifactorial nature involving barrier dysfunction, Th2-mediated immune responses, and chronic hypersensitivity.

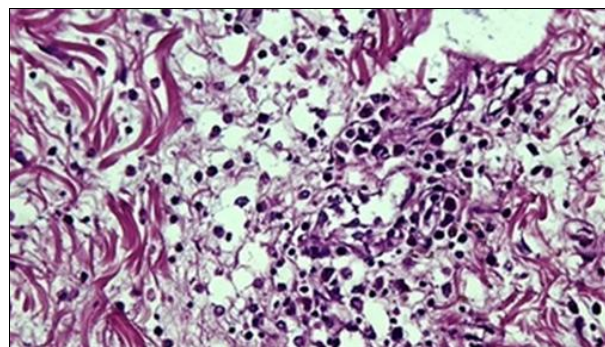


Fig 1: multi focal infiltration of loosely arranged mast cells with minimal cellularity along with eosinophils.

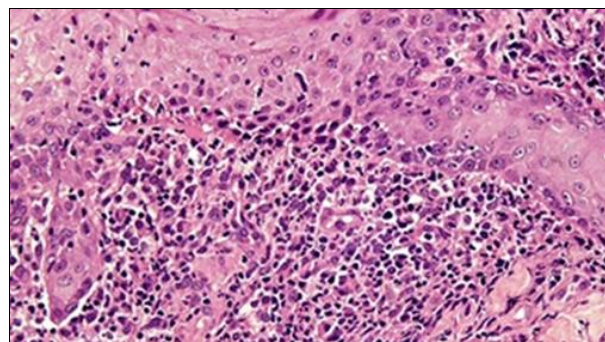


Fig 3: Lobular area surrounded by fibrous connective tissue showed formation of sweat glands with mild to moderate atypia forming central lumen containing secretions occasionally.

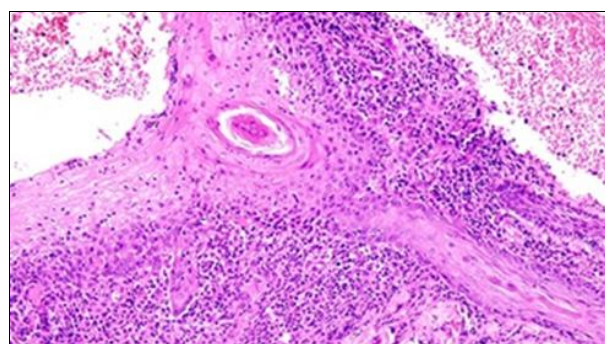


Fig 2: Diffuse infiltration of lymphocytes around the periphery of sweat glands and hair follicles

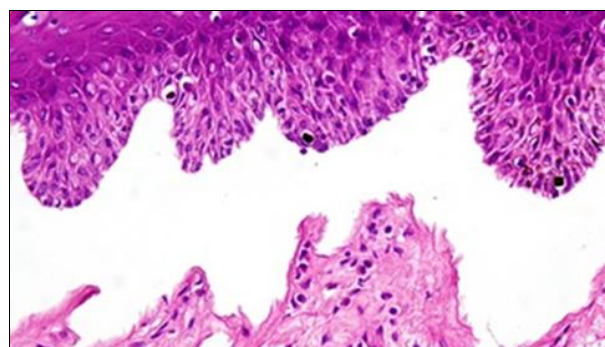


Fig 4: Complete separation of epidermis from dermis. The epidermis showed spongiosis with apoptotic squamous epithelium.

Discussion

The histopathological findings observed across six dogs with clinically confirmed canine atopic dermatitis (CAD) reveal a diverse yet consistent pattern of immune-mediated skin pathology. These changes reflect the chronicity, hypersensitivity, and barrier dysfunction that define CAD, and they align with the immunopathological mechanisms

described extensively in the literature (DeBoer & Hillier, 2001; Leung *et al.*, 2004; Marsella, 2021) ^[1, 7, 8].

One of the hallmark features observed was epidermal spongiosis, indicative of intercellular edema and early inflammatory insult. This was often accompanied by apoptotic squamous epithelium, suggesting cytokine-induced keratinocyte damage, particularly mediated by IL-31 and IL-4 (Dillon *et al.*, 2004; Santoro *et al.*, 2020) ^[3, 12]. In some cases, bullae formation at the dermal-epidermal junction and complete epidermal separation were noted, mimicking autoimmune blistering disorders such as bullous pemphigoid. While rare in CAD, such findings may represent severe barrier compromise or overlapping immunological phenomena (Robert & Kupper, 1999) ^[11].

The dermal compartment consistently showed infiltration of eosinophils, lymphocytes, plasma cells, and mast cells, reflecting a Th2-skewed immune response. Eosinophils, known for their cytotoxic granules, contribute to tissue damage and pruritus, while lymphocytes and plasma cells indicate chronic antigenic stimulation (Kim *et al.*, 2015; Eisenschenk *et al.*, 2024) ^[6, 4]. The presence of mast cells with anisocytosis, anisokaryosis, and nuclear pleomorphism supports the role of mast cell activation in CAD pathogenesis, consistent with findings by Neis *et al.* (2006) ^[9] and Santoro *et al.* (2020) ^[12]. These cells release histamine and other mediators that perpetuate itch and inflammation, particularly in response to IL-31 signaling. A notable observation was peri-follicular lymphocytic infiltration and lymphocytic hidradenitis, especially around sweat glands and hair follicles. This pattern is characteristic of chronic CAD and reflects persistent allergen exposure and immune activation at adnexal sites (Scott *et al.*, 1995; Nuttall *et al.*, 2013) ^[13, 10]. In some specimens, benign hyperplastic proliferation of the epidermis with acanthosis was seen, indicating chronic irritation and epidermal remodeling.

Importantly, no evidence of neoplasia was found in any sample, reinforcing the non-neoplastic nature of CAD despite its aggressive clinical presentation. These findings validate the utility of histopathology in CAD diagnosis—not only for confirming clinical impressions but also for identifying concurrent or mimicking conditions such as autoimmune dermatoses or chronic folliculitis.

Conclusion

The histopathological evaluation of six dogs clinically diagnosed with canine atopic dermatitis revealed a consistent pattern of epidermal disruption, immune cell infiltration, and adnexal inflammation, underscoring the multifactorial nature of the disease. Key features such as spongiosis, apoptotic keratinocytes, mast cell activation, and peri-follicular lymphocytic infiltration reflect the chronic hypersensitivity and Th2-dominant immune response central to CAD pathogenesis. The presence of bullae and epidermal separation in select cases suggests severe barrier compromise or overlapping autoimmune processes, while benign epidermal hyperplasia and hidradenitis indicate chronicity and follicular involvement.

These findings reinforce the diagnostic value of histopathology in CAD—not only for confirming clinical impressions but also for identifying concurrent or mimicking conditions. When integrated with clinical scoring systems and immunological profiling, histopathology enhances diagnostic precision and informs therapeutic decisions. As targeted biologics such as Lokivetmab gain prominence, understanding the tissue-level dynamics of CAD remains essential for optimizing patient outcomes and tailoring long-

term management strategies.

Conflict of Interest

Not available

Financial Support

Not available

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