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Diagnostic Exercise
From the Latin Comparative Pathology Group and the Davis-Thompson Foundation

Chronic exfoliative dermatitis in a German Shorthaired Pointer dog

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History: A 5-year-old, spayed female, German Shorthaired Pointer dog presented to a veterinary clinic for a second opinion following a history of chronic dermatitis and suspected allergies. Clinical signs improved with prednisone, but there was no appreciable response with antibiotics or oclacitinib (Apoquel). Lesions initially developed on the chest and occasionally involved paw pads and lips.

Clinical findings:

Upon presentation, crusts were noted at the oral mucocutaneous junctions, and marked crusting with follicular casts were noted on the trunk, most prominently on the ventrum. The ventrum exhibited coalescing areas of hyperpigmentation. Lesions progressed to generalized scale and crusts over the entire body (Figure 1). Complete blood count and serum chemistry results were within normal limits. The dog began to demonstrate a hunched stance while standing and a mild shifting leg lameness. Skin biopsies were submitted to a dermatopathology specialty service.

Follow-up questions:

- *Morphologic diagnosis:*
- *Name the disease:*
- *Etiology:*
- *Other affected organs:*

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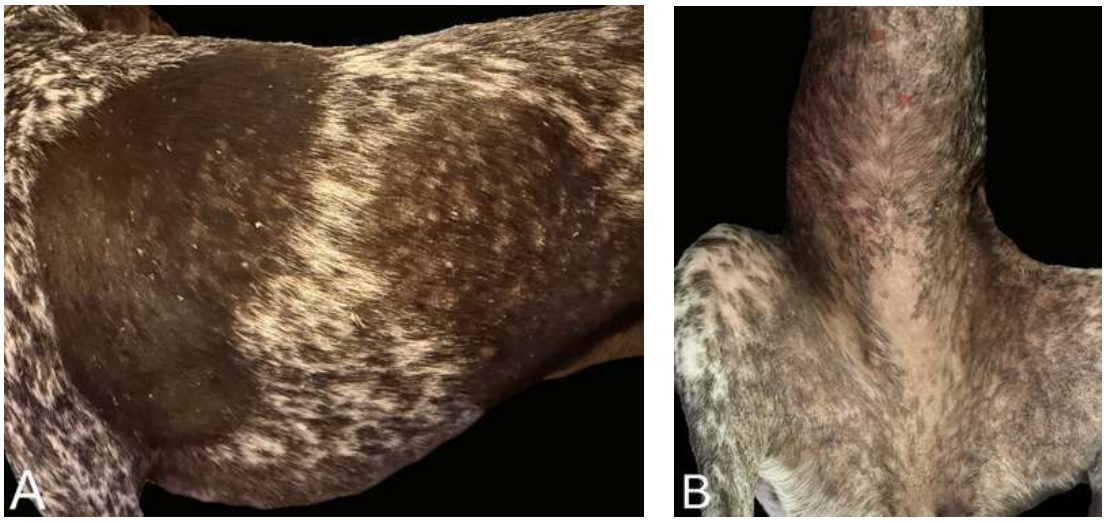

Figure 1. Photographs of the lateral trunk (A) and ventrum (B) of a 5-year-old spayed female German Shorthaired Pointer dog with chronic exfoliative dermatitis. The skin exhibits diffuse scaling and follicular casting with patchy areas of hypotrichosis to alopecia. Follicular casting is more prominent on the lateral trunk, while adherent scale is observed on the ventral skin.

ANSWERS

Histologic findings:

Three punch biopsies from the ventral and lateral trunk were fixed in neutral-buffered 10% formalin, bisected, and routinely processed for histologic evaluation with H&E staining. The epidermis was of normal thickness to mildly hyperplastic, and the surface was covered by moderate, basket-weave orthokeratotic hyperkeratosis that was occasionally intermixed with parakeratotic keratin and small numbers of degenerate neutrophils. Multifocally infiltrating the epidermis and frequently infiltrating hair follicle infundibula were moderate numbers of lymphocytes which multifocally obscured the dermoepidermal junction (Figure 2). There was frequent vacuolation and apoptosis of basal keratinocytes in both the epidermis and hair follicle infundibula. Less often, apoptotic keratinocytes were noted in the stratum spinosum (Figure 2). Occasionally, small subepidermal clefts were noted. Small numbers of macrophages in the superficial dermis contained melanin granules (pigmentary incontinence). Multifocally surrounding adnexal units were small to moderate numbers of plasma cells. Sebaceous glands were diffusely, markedly reduced in number and were completely absent in two of the biopsies.

Morphologic diagnosis: Moderate, multifocal, chronic, lymphocytic interface dermatitis and mural folliculitis with basal keratinocyte apoptosis, hyperkeratosis, and loss of sebaceous glands.

Name of the disease: Exfoliative Cutaneous Lupus Erythematosus (ECLE).

Etiology: Genetic. Genomic sequencing of German Shorthaired Pointers affected by ECLE identified homozygous mutant alleles in the UNC93B1 gene

Other affected organs: Joints, lymph nodes, kidneys.

Discussion: Exfoliative Cutaneous Lupus Erythematosus is a breed-specific form of Cutaneous Lupus Erythematosus (CLE) that has been predominantly reported in young German Shorthaired Pointers (1-5). Genomic sequencing of GSHPs affected by ECLE identified homozygous mutant alleles in the UNC93B1 gene, which leads to upregulation of Toll-like receptor 7 (TLR7) signaling with subsequent development of autoimmune disease (3, 4). Related breeds, including German Longhaired Pointers and Vizslas, carry the same mutant allele in the heterozygous state, strongly supporting a hereditary origin (4). Interestingly, two reported cases of mixed-breed dogs affected by ECLE carried the mutant gene in a homozygous state, which show how the pathogenic allele can be passed through mixed-breed dogs as well (3).

The most prominent skin lesions reported are scaling and crusting, sometimes with keratin fronds surrounding and entrapping hair shafts (follicular casts) (1-5). Lesions typically begin on the head and back before becoming generalized, and they may progress to erythema, hair loss, pigment alteration, and erosions and ulcers, with common secondary bacterial infection (1-5) As the skin lesions progress, affected dogs develop progressive lethargy and arthralgia, manifested as lameness and a hunched stance. Joint aspirates, spinal cord radiographs, myelogram, and cerebrospinal fluid analysis have been performed to investigate the cause of lameness and conformation, but these studies have not revealed any underlying abnormality (1, 2, 5). Other reported findings in dogs with ECLE include infertility, lymphadenomegaly,

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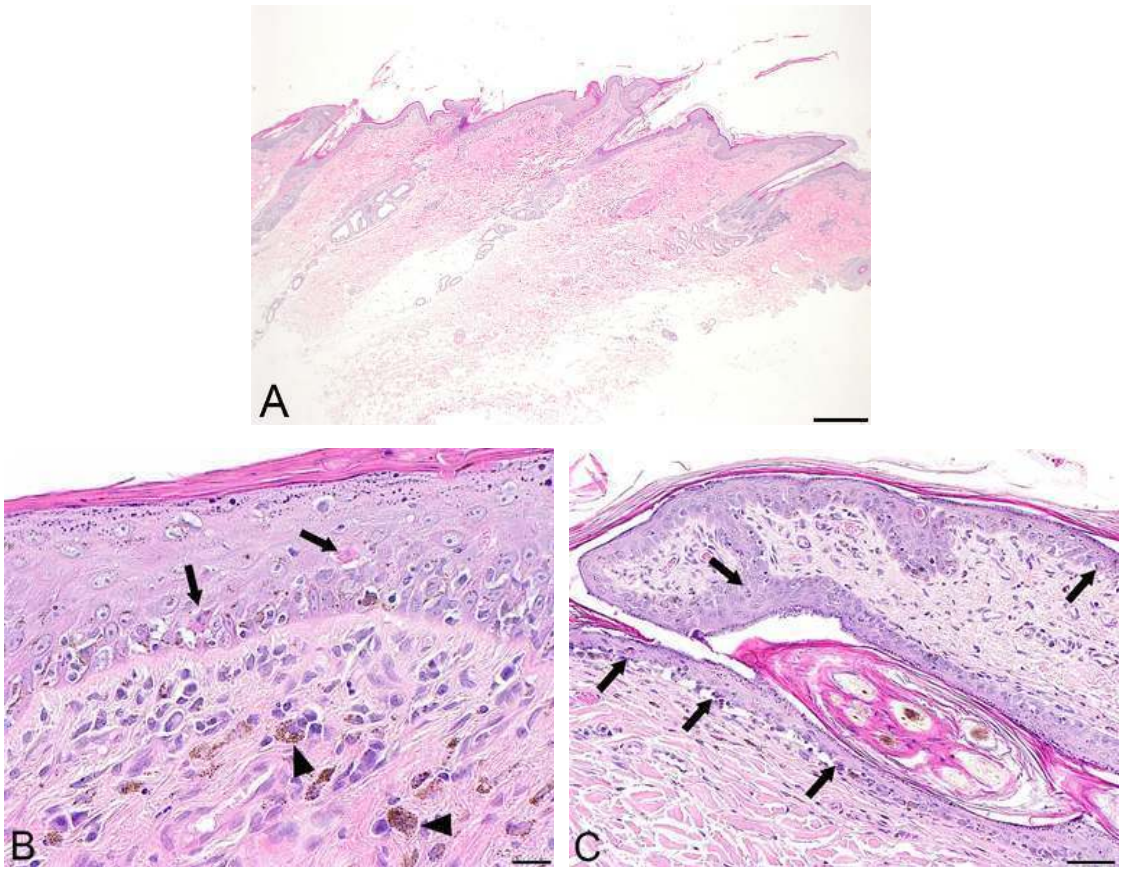

Figure 2. Photomicrographs of tissue sections from biopsy samples of the cutaneous lesions of the dog described in Figure 1. A – Moderate orthokeratotic and parakeratotic hyperkeratosis cover the skin surface. Hair follicles are also mild to moderately distended by keratin (follicular keratosis), which surrounds hair shafts. H&E stain; bar = 500 µm. B – Higher magnification of the epidermis showing an interface dermatitis with infiltration of the dermoepidermal junction by lymphocytes, apoptosis of keratinocytes (arrows), and pigmentary incontinence in the superficial dermis evidenced by macrophages containing melanin granules (arrowheads). H&E stain; bar = 20 µm. C – Interface mural folliculitis is also observed with numerous apoptotic keratinocytes (arrows) predominantly distributed in the basal cell layer. The interface dermatitis/folliculitis in this case has resulted in cleft formation between the dermis and epidermis. H&E stain; bar = 50 µm

hematological abnormalities (lymphopenia, thrombocytopenia), and glomerulonephropathy (1, 2, 3, 5). Dogs with the immune complex membranous glomerulonephropathy are not azotemic but frequently exhibit proteinuria and an elevated urine protein-to-creatinine (UPC) ratio (1). The glomerulonephropathy is hypothesized to be a manifestation of systemic lupus erythematosus, making ECLE unique amongst the cutaneous lupus subtypes in dogs, which do not demonstrate systemic involvement (1).

The most characteristic histologic lesions are lymphocyte-rich interface dermatitis with marked hyperkeratosis, superficial mural folliculitis, and vacuolar degeneration of the basal layer of the epidermis accompanied by apoptosis (1-5). Pigmentary incontinence is frequently observed in the superficial dermis, and results from basal keratinocyte damage. Keratinocyte vacuolation and apoptosis also prominently affect hair follicle infundibula to the level of the adnexal glands, and marked follicular and sebaceous gland atrophy may be present (1, 2, 3, 5)

Differential diagnoses include other forms of cutaneous lupus erythematosus, sebaceous adenitis, and erythema multiforme (1,5). Sebaceous atrophy or loss observed in ECLE may resemble sebaceous adenitis, but sebaceous adenitis is not characterized by interface dermatitis and folliculitis. Discoid Lupus Erythematosus (DLE) is usually restricted to the face and histologically presents a more intense lichenoid interface dermatitis and less marked hyperkeratosis than ECLE, although basal cell degeneration and basement membrane blurring may be similar in both diseases. In Erythema Multiforme (EM), apoptotic keratinocytes are found at all levels of the epidermis and are accompanied by lymphocyte satellitosis. In contrast, ECLE shows keratinocyte apoptosis primarily in the basal layer, although individual necrotic keratinocytes can occasionally occur at all epidermal levels, making it difficult to distinguish it from EM (2). Knowledge of the affected breed and the age of onset can help distinguish

ECLE from other forms of cutaneous lupus erythematosus as well as drug reactions (2).

There is no effective treatment for ECLE. Reported cases show only subjective, transient improvement, with continued disease progression despite therapy (5). Dogs with ECLE are typically euthanized due to disease progression or adverse effects associated with medication. This makes ECLE the most challenging CLE variant to treat (1, 3, 5).

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