History

Three related Persians (sire and 2 kittens [1 male and 1 female]) had a history of alopecia, hyperpigmentation, scales, crusts, and a general poor-quality coat. In the kittens, the condition was present since soon after birth. Despite broad-spectrum treatment (antimicrobials, glucocorticoids, antifungal drugs, and an oral fatty acid supplement) administered for a few months, the cutaneous lesions did not improve. At 5 months of age, the female kitten was evaluated because of the dermatologic lesions.

Clinical and Gross Findings

The kitten was alert, vaccinated, and dewormed and in good body condition. On physical examination, the skin was dry and the hair was of poor quality and dull. The animal had generalized hypotrichosis and hyperpigmentation without pruritus (Figure 1). Severe alopecia, scales, and crusts were present mostly in the periocular region and over the nose. No other clinical alterations were observed.

Formulate differential diagnoses from the history, clinical findings, and Figure 1—then turn the page→
Histopathological Findings

Multiple punch skin biopsy specimens were obtained from the kitten’s interscapular region. On histologic examination of sections of the skin biopsy specimens, diffuse atrophy of sebaceous glands, which had an undulating profile, was observed. The lobulated morphological characteristic of these structures was lost, and individual sebaceous lobules were poorly defined. Centripetal progression from basaloid cells to mature sebocytes was absent. Instead, discrete clusters of sebocytes and a higher number of small reserve cells were haphazardly arranged around the follicular isthmus (Figure 2). Remaining sebocytes contained variably sized vacuoles and intracytoplasmic eosinophilic inclusions (keratin). Shrunken hypereosinophilic sebocytes with pyknotic nuclei, reflecting cellular apoptosis, were also observed (Figure 3). Hair follicles had evidence of trichomalacia, and most of them were in anagen. Mild orthokeratotic hyperkeratosis was also observed.

Morphologic Diagnosis and Case Summary

Morphologic diagnosis and case summary: sebaceous gland dysplasia in a kitten.

Comments

Sebaceous gland dysplasia is a very rare congenital dermatosis in young dogs and cats and is associated with abnormal sebaceous gland formation and differentiation. Clinically, the disease is characterized by a poor-quality coat and progressive hypotrichosis, initially involving the head, pinnae, and dorsum.1–3 In the kitten of the present report, cutaneous lesions were generalized, but it is possible that the process had started on the head given the presence of secondary lesions (periocular crusts, scales, and severe alopecia) in that body region.

Because the condition is first noted in animals at a young age, suspicion of scabies, fungal skin disease, or a congenital cutaneous disorder may be raised. The former can be easily ruled out by microscopic examination of skin scrapings, direct examination of the hairs, or fungal culture of hair follicles. The most striking clinical sign associated with sebaceous gland dysplasia is nonpruritic hypotrichosis. This clinical finding points to a noninflammatory failure of hair growth, usually associated with endocrinopathies or congenital skin disorders. The absence of systemic clinical signs and results of appropriate serum analyses may promptly exclude endocrine disease.

Examination of skin biopsy specimens is necessary to confirm the diag-

Figure 2—Photomicrograph of a section of a skin biopsy specimen obtained from the interscapular region of the kitten in Figure 1. Irregular aggregates of haphazardly arranged basaloid epithelial cells (arrow) and atypical sebocytes (arrowhead) around a follicular isthmus are visible. H&E stain; bar = 100 µm.

Figure 3—Photomicrograph of the same section of a skin biopsy specimen obtained from the interscapular region of the kitten in Figure 1. Sebaceous glands are composed of atypical sebocytes that contain variably sized vacuoles and a large proportion of small reserve cells. Notice the keratin accumulation (arrowhead) and apoptosis (arrow). H&E stain; bar = 100 µm.
nosis of a congenital pilosebaceous disorder. Histologically, sebaceous gland dysplasia is characterized by atrophy and abnormal morphological features of the sebaceous glands, represented by a reduction or absence of normal sebocytes along with an increase of reserve cells lacking the typical lobular architecture.\textsuperscript{1,3} The histologic lesions are very distinctive; therefore, histologic diagnosis of sebaceous gland dysplasia is easy to achieve and there are not many differential diagnoses to consider. However, in chronic cases, when inflammatory cells infiltrate the follicular walls or the sebaceous glands, lymphocytic mural folliculitis or sebaceous adenitis should be suggested as differential diagnoses. In the case described in the present report, there was no noteworthy inflammatory infiltrate; thus, a diagnosis of sebaceous gland dysplasia was established.

Given that sebaceous gland dysplasia is a congenital disease affecting very young animals,\textsuperscript{1–3} some authors have suggested the possibility of a hereditary condition because some littermates have identical clinical findings.\textsuperscript{3} Along with the kitten of the present report, 2 closely related family members (its sire and a sibling) had similar cutaneous lesions with no other clinical problems, which reinforces the hypothesis of a hereditary disease.

The cause of sebaceous gland dysplasia in cats is unknown, but in utero exposure to toxins or viruses and poor-quality nutrition of the dam during pregnancy have been suggested as factors that can interfere with the skin appendage differentiation and maturation program.\textsuperscript{3} Sebaceous gland development is a complex process involving several signaling pathways, hormones, growth factors, and mediators of lipid metabolism.\textsuperscript{4} To date, there are no published studies regarding the genetic control of sebaceous gland development in felids, to our knowledge. However, in laboratory rodents, some failures in the control of lipid metabolism have been described and certain genes coding for enzymes and receptors involved in such processes, namely stearoyl-CoA desaturase and peroxisome proliferator-activated receptor-\(\gamma\) (PPAR-\(\gamma\)), have been identified.\textsuperscript{5,6}

Because this condition represents an abnormal process of sebaceous gland development and maturation, the potential for total recovery should not be presumed. However, clinical improvement of the cutaneous lesions has been described with topical medication (antiseborrheic shampoos and moisturizing conditioners) or oral supplementation with omega-6 and omega-3 fatty acids and vitamin A. Depending on the animal’s response, corticotherapy or immunosuppressant therapy may be needed.\textsuperscript{5,7} For the kitten of the present report, treatment included topical application of olive oil and use of antiseborrheic shampoos. The owners reported a slight improvement of the cutaneous lesions with this treatment regimen along with limited solar exposure. There was no available information about treatment or outcome for the other kitten and the sire.

References