CASE OF INTEREST

A case of pemphigus vulgaris in a dog.

By Richard Fox, Veterinary Pathologist

A 7 year old male entire Labrador presented with rapid onset multifocal to coalescing areas of ulceration affecting nasal planum, prepuce, lips, gingiva, soft and hard palate. Paroxysmal sneezing was also noted. Nasal flushes were performed but no significant changes were noted on rhinoscopy and cytological examination. Autoimmune disease was suspected clinically and biopsies were taken from preputial, nasal planum and oral cavity.

Three sections of tissue were examined, one identified oral mucosa, the other two haired skin. Two of the cutaneous lesions displayed ulceration but in the oral mucosal biopsy there was suprabasilar clefting of the mucosal epithelium.

Figure 1. Histological section of the oral biopsy displaying an ulcer and bordering mucosal epithelium with conditioned suprabasal clefting. HE Stain.

This also was present, as a conditioned artefact, in one piece of haired skin which affected hair follicles.

Figure 2. Histological section showing tombstoning of residual basal cells with acantholysis. HE Stain.

Occasional neutrophils were present in the cleft and there was also evidence of mild acantholysis in the areas of clefting. Areas of ulceration were evident adjacent to the areas of clefting.

Figure 3. Histological section of haired skin showing further suprabasilar epidermal clefting and ulceration. HE Stain.

A diagnosis of pemphigus vulgaris was made on the clinical and histopathological findings.

Pemphigus vulgaris (PV) is a very rare, severe vesicobullous and ulcerative autoimmune disease reported in the dog and cat. Desmoglein 3 (Dsg3), an adhesion molecule (cadherin) of keratinocytes, is more strongly expressed in the suprabasilar keratinocytes of the oral mucosa, and has been identified as the targeted antigen in canine PV, as occurs in humans. However, when lesions of PV affect the hairy skin as well as mucous membranes, other antigens such as desmoglein 1 (Dsg1), which is more strongly expressed in superficial keratinocytes of haired skin, may be targeted.

Drug-induced PV may occur in humans; removal of the offending drug leads to reversal of the lesions (Brenner er al., 1998). PV-like skin reactions putatively due to drug therapy (including sulfasalazine) have been observed sporadically in both dogs and cats. Also, transient, often orally confined, cases not explained by adverse drug reactions have been seen by the authors in these species.

The exact pathomechanism of vesicle and bulla formation is not known. Autoantibodies have been found but do not on their own give rise to lesions and investigation is ongoing.
Histologically the suprabasilar acantholysis creates fragile, transient vesicles and bullae that rapidly progress to ulcers. Partial bilateral symmetry, especially facial, can be striking. Fragile, irregularly shaped vesicles and bullae develop in groups. Erythema may precede vesicle formation, especially in hairless skin areas (Olivry, 2003). Rupture of vesicles and bullae rapidly leads to erosions; secondary surface bacterial overgrowth then results in widespread ulceration. Crusting is a feature of nonmucosal ulcerations. Ulcers expand, creating lesions that are substantially larger than the previously intact bullae. These ulcers are irregularly shaped and may coalesce further. A positive Nikolsky sign (the artificial extension of a blister or ulcer induced by digital pressure to adjacent mucous membrane or skin) may be elicited.

Most dogs and cats have lesions affecting the oral cavity and mucocutaneous junctions. Coalescing ulcers on the tongue palate, and gingiva which are not contiguous with the teeth should increase clinical suspicion for PV. Lip involvement usually is seen with extensive oral disease. Thick,ropy,tenacious,odorous saliva is an additional feature.

A small number of cases present as a therapeutic challenge. Often these have been on traditional modes of therapy, and have either failed to respond or have had adverse reactions to these forms of therapy. Switching the type of glucocorticoid therapy may obtain a response. If this fails, trying aggressive glucocorticoid shock intravenous therapy can also be of value. This

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