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Glossitis in dogs arises from diverse etiologies but shows predominantly chronic ulcerative pathology and low clinical detection rates

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Objective

To describe the clinical and histopathological features of glossitis in dogs and determine its clinical-pathological correlation.

Methods

Medical records and pathology reports of dogs diagnosed with glossitis at the William R. Pritchard Veterinary Medical Teaching Hospital, University of California-Davis, were reviewed (2009 to 2024). Dogs diagnosed with glossitis that was confirmed histologically were included in the study. Dogs that had concurrent histological diagnosis of lingual neoplasia were excluded.

Results

100 dogs were included. Awake oral examination failed to detect 55% of the glossitis lesions. Six inciting causes of glossitis were identified, with trauma and idiopathy being the most common causes, followed by inflammatory causes (associated with concurrent local oral or dental disease), oral manifestations of systemic disease (other than immune-mediated or autoimmune etiology), immune-mediated or autoimmune causes, and foreign body. Pooled data revealed that glossitis commonly presented as either a unilateral or bilateral solitary lesion, affecting the ventral surface of the middle or rostral third of the tongue. The predominant histological features were chronic, severe ulcerative or erosive neutrophilic inflammation, myodegeneration, and fibrosis.

Conclusions

Glossitis may be presented as a primary disease or manifestation of underlying systemic conditions. The appearance and presentation of different causes of glossitis can be similar; hence, additional clinical information and ancillary testing should be applied to determine the pathogenesis.

Clinical Relevance

This study emphasizes the importance of integration of clinical-pathological data in a multidisciplinary approach for accurate diagnosis and proper treatment planning to optimize clinical outcomes.

Keywords: glossitis, dogs, clinical-pathological correlation, tongue, lingual

The tongue is a muscular structure lined by keratinized epithelium on its dorsal surface and nonkeratinized epithelium on its ventral surface. In addition, the dorsal keratinized surface of the tongue is considered masticatory mucosa and characterized by specialized filiform, fungiform, vallate, foliate, and conical papilla, which have a role in taste and tactile sensation. The tongue plays an essential role in

prehending, masticating, and swallowing food; lapping water; nursing; grooming; and dissipating heat in dogs. Dogs with lingual lesions may present with difficulty swallowing and prehending food, regurgitation, hypersalivation, abnormal positioning of the tongue, and pain, depending on the lesion's nature and extent.^{1,2} The most reported histopathological diagnosis of lingual lesions in dogs is neoplasia, followed by inflammatory lesions or glossitis and others, including calcinosis circumscripta.^{3,4}

Glossitis accounted for about one-third of the lingual lesions in dogs in a large retrospective study based on histology.^{3,4} Proposed causes of glossitis include infection, chemical or mechanical injury, electrocution, or migrating foreign body,^{5,6} which

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can be linked to the reported history. Other underlying systemic medical conditions, such as metabolic, infectious, immune-mediated or autoimmune, and paraneoplastic disease, can also be associated with glossitis or oral mucosal lesions in dogs.⁵⁻⁸ However, the inciting cause of glossitis may not be revealed with histology alone.^{3,4}

The clinical data on glossitis in dogs remain sparse and are mainly limited to case reports.^{1,2,9} Glossitis has been described as a change in the surface appearance of the tongue, such as a change of texture or color, loss of lingual papillae on the dorsal surface tongue,¹⁰ or the presence of nodule(s)⁹ or hyperplastic lesion(s). In human medicine, glossitis exists in different clinical patterns: atrophic, geographic, strawberry, median rhomboid, geometric, and benign migratory, correlating to the specific etiology or inciting cause of the disease.¹¹ In dogs, little is known about the relationship between the clinical characteristics of glossitis and its etiology and pathology. The present descriptive study aimed to summarize and illustrate the presentation, clinical features, and histopathological findings of glossitis in dogs and determine the clinical-pathological correlation of this disease entity, taking both clinical and histopathological findings into account. This study emphasized the importance of integrating clinical-pathological data to obtain an accurate diagnosis and thereby optimize clinical outcomes.

Methods

Data collection

The medical record database of the William R. Pritchard Veterinary Medical Teaching Hospital, University of California-Davis, was searched for dogs diagnosed with glossitis between January 1, 2009, and December 31, 2024, with use of the keyword *glossitis*. The medical records and pathology reports of those dogs were evaluated. Dogs diagnosed with glossitis by gross appearance alone (without a biopsy and histopathology) or those with a concurrent histological diagnosis of lingual neoplasia were excluded from the study. Data retrieved from the medical records included signalment, presenting complaints, service to which the patient was initially presented, history of comorbidities, history of exposure to chemicals or toxins, and oral examination findings. Dental charting was reviewed when available. Clinical descriptions of the lingual lesion were documented, including its distribution (unilateral, bilateral, or midline), pattern (solitary, multifocal, or diffuse), location (rostral or middle third or caudal third), surface (dorsal, ventral, or lateral), size, color, shape, and appearance. Information was noted of other mucosal lesion(s) present in the oral cavity. Imaging modality and biopsy method adopted for the lesion were also noted, along with the results of bacterial culture and sensitivity test, when available. Data retrieved from the pathology report included the microscopic descriptions of the lesion and histopathological diagnosis. Information was recorded related to the se-

verity (minimal, mild, moderate, or severe, depending on the density of inflammatory cells and extent of architecture changes), duration and onset, type of cell infiltration, and other histological features of the glossitis lesion. Finally, treatment and follow-up evaluation were documented.

Statistical analysis

Descriptive statistics were calculated. The type of distribution was identified with the Kolmogorov-Smirnov test of normality. Data that were normally distributed are presented as mean $\pm$ SD. Data that were not normally distributed are presented as median values, along with IQR. Categorical data are expressed as frequencies and percentages.

Results

Signalment and clinical presentation

A total of 100 dogs of 45 breeds met the inclusion criteria for the study. Eighty-four dogs (84%) were pure breeds, and 16 dogs (16%) were cross-breeds. The median (IQR) age at the time of diagnosis of glossitis was 9 years (5 to 11 years). There were 48 castrated male dogs (48%), 40 spayed female dogs (40%), 8 intact male dogs (8%), and 4 intact female dogs (4%). The presenting complaint of 19 dogs (19%) was lingual lesion or abnormality (which was later diagnosed as glossitis), while 81 dogs (81%) were presented to the veterinary medical teaching hospital for other illnesses. Thirty-eight dogs (38%) were reported to show various signs of dysphagia, including increased difficulty or inability to prehend food and increasing time needed for eating and drinking ($n = 19$); other reported signs were ptyalism (12), lip smacking or abnormal and excessive tongue movement (6), preference for soft food over dry food (5), reluctance to chew on hard toys or loss of interest in playing with toys (2), abnormal jaw movement or jaw chattering (2), and hesitation to eat (1). Ten of those 38 dogs (26.3%) showed > 1 sign listed prior. Owners of 61 dogs (61%) did not report signs or behavioral changes that were related to abnormality in the oral cavity. The remaining dog (1%) had no detailed history about the chief complaint, as this dog was presented for humane euthanasia. Most dogs were initially presented to the dentistry and oral surgery service (42 of 100 [42%]), followed by the internal medicine service (25 of 100 [25%]) and emergency service (12 of 100 [12%]). Twenty-one dogs were presented to other services, including neurology, surgery, community, dermatology, and oncology. Owners of over half of the cases (56 of 100 [56%]) specifically reported that their dogs had no exposure to toxins or chemicals.

Medical history and dental assessment

Comorbidities other than dental and oral disease were reported in 74 dogs (74%); 22 of 74 dogs (29.7%) had > 1 comorbidity (**Table 1**). Medication history was obtained for 62 dogs (62%). Sixty cases (96.8%) had ongoing medication courses, and medications were classified as antibiotics, NSAIDs, and

Table 1—Comorbidities and dental assessment of dogs with glossitis.

Comorbidity	No. of dogs
Other than dental and oral disease (n = 74)	
Cardiovascular disease	4
Dermatological disease	14
Gastrointestinal disease	10
Hematological disease	6
Metabolic disease	6
Neurological disease	11
Ophthalmological disease	4
Orthopedic disease	12
Respiratory disease	10
Reproductive disease	4
Renal disease	8
Urinary tract disease	5
Dental and oral disease with assessment (n = 34)	
Occlusion status (n = 27)	
Normocclusion	21
Class I malocclusion	1
Class II malocclusion	1
Class III malocclusion	4
Class IV malocclusion	0
Plaque index (n = 25)	
1	5
2	8
3	10
4	2
Calculus index (n = 25)	
0	2
1	4
2	8
3	9
4	2
Periodontal disease (n = 31)	
Stage 1	5
Stage 2	7
Stage 3	3
Stage 4	16
Dentoalveolar trauma	14
Attrition/abrasion	9

pain medications. Drugs of other classes included anticonvulsant, antihypertensive, orexigenic, gastro-protectant, proton pump inhibitor, insulin, thyroid, immunomodulatory, and cardiac medications, as well as renal or hepatic supplements. Dental charts were available for review in 34 cases (34%). Common dental pathology described in the assessment (taking dental radiographic findings into account) included periodontal disease, dentoalveolar trauma, and tooth abrasion or attrition.

Clinical characteristics of glossitis lesions

The size of the lingual lesions was recorded in 41 dogs (41%), with the distance across the lesion ranging from 2 to 60 mm (mean $\pm$ SD, 14.8 $\pm$ 11.8 mm). Various clinical descriptions of the lesions were noted. Their appearances were described as ulcerative, proliferative, lobulated, necrotic, erythematous, mottled, thickened, raised (border), depressed, mass-like, tubular swelling, mucosal enlargement, plaque-like, ridges with teeth marks, or loss of filiform papilla. The different shapes observed included round, nodular, irregular, linear, or

pedunculated. The color was noted as pale, white, or erythematous. The texture was described as either soft or firm.

The distribution and pattern were described in 90 dogs (90%). Unilateral and bilateral involvement were each present in 40 cases (44.5%), and midline glossitis was observed in 10 cases (11.0%). The affected side was recorded in 29 dogs (72.5%) with unilateral distribution of glossitis; the left side (19 of 29 [65.5%]) was more commonly affected than the right side (10 of 29 [34.5%]). Glossitis was more frequently seen as a solitary lesion (52 of 90 [57.8%]), followed by multifocal lesions (23 of 90 [25.5%]) and diffuse lesions (15 of 90 [16.7%]).

The surface of the tongue affected was described in 79 dogs (79%). Glossitis affected 1 surface (56 of 79 [70.9%]) more commonly than 2 surfaces (18 of 79 [22.8%]) or 3 surfaces (5 of 79 [6.3%]). When glossitis was identified on 1 surface, the ventral surface was most frequently affected (42 of 56 [75%]), followed by the lateral surface or border (8 of 56 [14.3%]) and dorsal surface (6 of 56 [10.7%]).

The location (rostral, middle, or caudal third) of glossitis was recorded in 46 dogs (46%). Of those, 37 lesions affected only 1 site, 8 lesions affected 2 sites, and only 1 lesion had 3 sites involved. When glossitis involved a single site, the rostral third (14 of 37 [37.8%]) and middle third (14 of 37 [37.8%]) of the tongue were equally affected in terms of frequency; 9 cases (24.4%) had only the caudal third of the tongue involved.

Evaluation and diagnostic methods

Lingual lesion(s) were identified in 41 dogs (41%) during the awake clinical examination; 55 dogs (55%) had the tongue lesion(s) detected at sedated or anesthetized oral examination or necropsy. Four dogs (4%) had no such details specified on the oral examination. Sixty-seven dogs (67%) had glossitis diagnosed via biopsy when alive; 33 dogs (33%) had glossitis diagnosed during necropsy. Biopsy method was described in 62 of the 67 dogs (92.5%). Incisional biopsy and excisional biopsy were performed in 52 of 62 dogs (83.9%) and 10 of 62 dogs (16.1%), respectively. Among those with incisional biopsy performed, biopsy punch was used in 14 cases (14 of 52 [26.9%]). Imaging modality was adopted in 10 dogs (10%), but only half of the cases had the primary intention to investigate the lingual lesion. In total, 9 dogs received CT scan of the skull (with contrast agent) and 1 dog had an ultrasound scan on the lingual lesion in addition to the CT scan. One dog had an MRI of the head performed. The location and extent of the lingual lesion were usually well described from the CT scan, with common features described as contrast-enhancing, presence of hypoattenuating tract, irregular, large, or nodular. Ultrasound identified hypoechoic change and linear striations in the lingual lesion that were interpreted as hair for the latter. Magnetic resonance imaging description of the lingual changes was nonspecific, with a description of a well-defined, gas-filled cavitation at the masti-

catory muscle that immediately abutted the tongue. Aerobic and anaerobic culture and sensitivity tests of the lingual lesion were tested in 4 dogs (4%). Microbes that were identified from aerobic culture included *Pasteurella canis*, coagulase-negative staphylococci, α -hemolytic streptococcus, *Pseudomonas aeruginosa*, *Streptococcus dysgalactiae* subsp *equisimilis*, *Staphylococcus aureus*, *Staphylococcus pseudintermedius*, nonhemolytic *Escherichia coli*, *Mycoplasma* sp, *Pasteurella dagmatis*, *Streptococcus canis*, and viridans group streptococci. Microbes that were detected from anaerobic culture included *Fusobacterium nucleatum*, *Peptostreptococcus anaerobius*, *Bacteroides fragilis*, and anaerobic gram-positive rods.

Histopathological findings

All cases underwent histopathological evaluation of the lingual tissue; histopathological diagnosis of glossitis was confirmed. Various degrees of severity of glossitis were specified in 87 cases, of which 4 cases (4.6%) were described as minimal with few inflammatory cells and normal tissue architecture, 12 cases (13.8%) as mild with scattered inflammatory cells, 23 cases (26.4%) as moderate with a higher number of inflammatory cells, and 48 cases (55.2%) as severe with heavy infiltration of inflammatory cells and marked architecture distortion. The duration and onset of the glossitis were evaluated in 48 cases, of which 5 (10.4%) were reported as subacute, 14 (29.2%) as acute, and 29 (60.4%) as chronic. Involvement of various cellular components was reported in 74 cases, of which 19 (25.7%) were reported with infiltration of only 1 cell type and 55 (74.3%) had mixed cell types. Neutrophilic infiltration was most commonly seen ($n = 48$), followed by lymphoplasmacytic (29), histiocytic (16), plasmacytic only (5), lymphocytic only (3), eosinophilic (2), and mastocytic infiltration (2). Half of the cases (50 of 100 [50%]) had glossitis assessed as ulcerative or erosive. Other histological evaluations included necrosis (30 of 100 [30%]), suppurative (14 of 100 [14%]), granulomatous or presence of histiocytic infiltration (14 of 100 [14%]), pyogranulomatous (2 of 100 [2%]), presence of fibrosis (5 of 100 [5%]), and proliferative (3 of 100 [3%]), with 1 case that specified proliferation of spindle cells. Intralesional bacteria (10 of 100 [10%]), mineralization (5 of 100 [5%]), foreign object (4 of 100 [4%]), and hair shafts (4 of 100 [4%]) were identified. Epithelial dysplasia was diagnosed in 5 cases (5%).

Clinical-pathological correlations

The correlation of clinical assessment and histological diagnosis revealed 6 inciting causes in the studied population: traumatic (30 of 100 [30%]), idiopathic (30 of 100 [30%]), inflammatory (associated with concurrent local oral or dental disease; 14 of 100 [14%]), manifestation of systemic disease other than immune-mediated or autoimmune etiology (13 of 100 [13%]), immune-mediated or autoimmune (8 of 100 [8%]), and related to a foreign body (5 of 100 [5%]). The clinical

and histopathological data according to each category of inciting cause were further stratified and are presented in **Supplementary Table S1**. The clinical appearance of various causes of glossitis with corresponding histology images is illustrated in **Figures 1 to 4**. Additional examples of clinical presentation of glossitis of specific etiology are shown in **Figure 5**.

Traumatic glossitis (30 of 100 [30%]) was deemed to be related to mechanical injury (either self [25 of 30 {83.3%}] or external [3 of 30 {10%}]) or chemical injury (2 of 30 [6.7%]). Self-trauma was most reported and happened when tissues were trapped between the teeth during chewing or seizure episodes. The glossitis was described mostly as unilateral, solitary, affecting the ventral surface of the tongue, and occurring more commonly at either the middle or caudal third of the tongue. Histologically, traumatic glossitis was mostly identified as chronically, severely inflamed, with frequent neutrophilic inflammation, and often described as ulcerative or erosive. The presence of necrotizing or granulation tissues was also reported. Intralesional bacteria, hair shaft(s), or both were uncommonly seen. Epithelial dysplasia was detected, but it was rare.

The diagnosis of idiopathic glossitis (30 of 100 [30%]) was reached when both clinical assessment and histology were unable to identify a definitive cause of the glossitis. Glossitis of this type was most frequently reported as unilateral, solitary, affecting the ventral surface of the tongue, and more commonly seen at the rostral or middle third of the tongue. Histologically, glossitis of this type was mostly identified as chronic and severely inflamed, with frequent neutrophilic or lymphoplasmacytic inflammation or both, and often described as ulcerative or erosive. Histiocytic infiltration was also reported in some cases. Other histological features varied greatly in this type of glossitis, with the descriptions including necrotizing; suppurative; with granulation tissues, mineralization, or fibrosis; proliferative; or pyogranulomatous. Epithelial dysplasia was rarely noted.

Glossitis of inflammatory cause (14 of 100 [14%]) was deemed to be associated with inflammatory disease happening concurrently in the oral cavity, namely periodontal disease (12 of 14 [85.7%]) and canine chronic ulcerative stomatitis (2 of 14 [14.3%]), likely due to the close contact of the lingual mucosal tissues with dental plaque and calculus or as part of the disease process for the latter. This type of glossitis was most frequently reported as bilateral, multifocal, or diffuse, affecting the ventral surface of the tongue and more commonly seen at the middle third. Histologically, glossitis of this type was mostly identified as chronic and severely inflamed, with frequent neutrophilic or lymphoplasmacytic inflammation or both, and often grossly described as ulcerative, erosive, or suppurative.

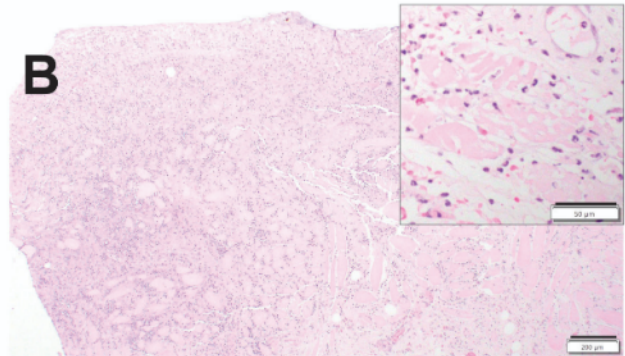
Cases of glossitis as oral manifestations of systemic disease (other than immune-mediated or autoimmune etiology; 13 of 100 [13%]) in this cohort were

TRAUMA

EARLY



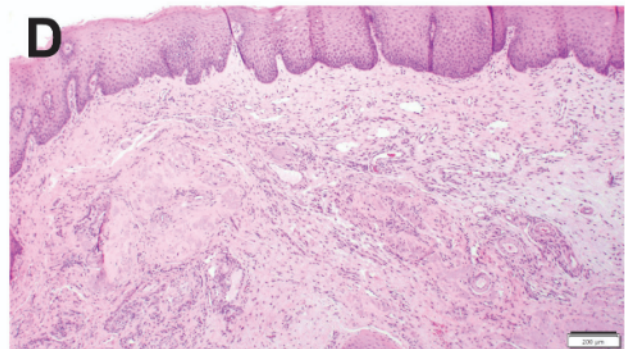
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LATE



D



FOREIGN BODY

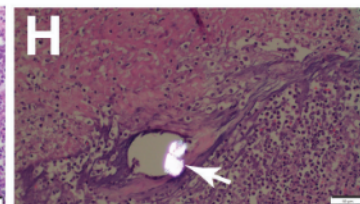
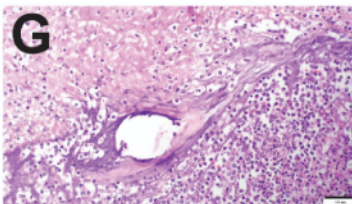
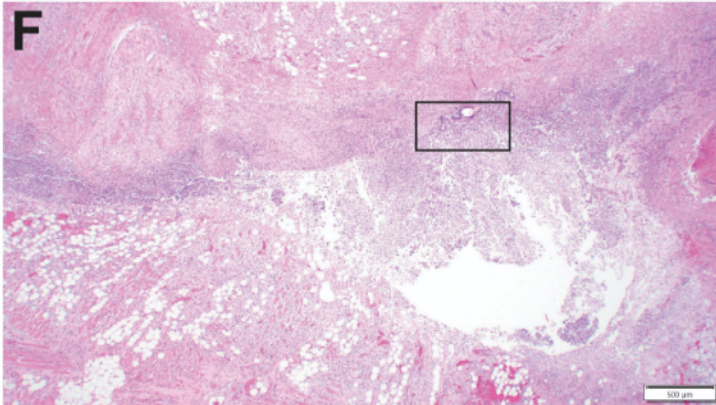


Figure 1—Clinical appearance of traumatic glossitis on the right ventral surface of the tongue in the early stage (A) and late stage with the presence of a raised and proliferative lesion (C). B—Low and high magnification of the tongue biopsy demonstrating extensive myonecrosis with neutrophilic inflammation. The inflammatory cells (primarily neutrophils) are expanding the endomysial space. The sarcolemma of individual myofibers is fragmented, and there is loss of cross striations. Bar = 200 µm (inset bar = 50 µm); H&E stain. D—Histological presentation of the same lesion shown in panel C. Note that the lingual musculature healed by fibrosis and minimal inflammatory infiltration is present. The surface epithelium is fully regenerated. Bar = 200 µm; H&E stain. E—Clinical appearance of glossitis associated with a foreign body. Note the discoloration of the rostral half of the tongue and the presence of a white, linear stand of material at the dorsal aspect of the caudal root of the tongue. F—The histopathology of the same lesion shown in panel E reveals necrosuppurative mucositis and myositis. Bar = 500 µm; H&E stain. G—Higher magnification on the suspect foreign body enclosed in the black rectangle in panel F. Note the associated neutrophilic cellular infiltration and fibrin exudation. Bar = 100 µm; H&E stain. H—Same section as that shown in panel G, illuminated by a polarized light. Note the strong birefringence of the foreign material (arrow). Bar = 100 µm; H&E stain.

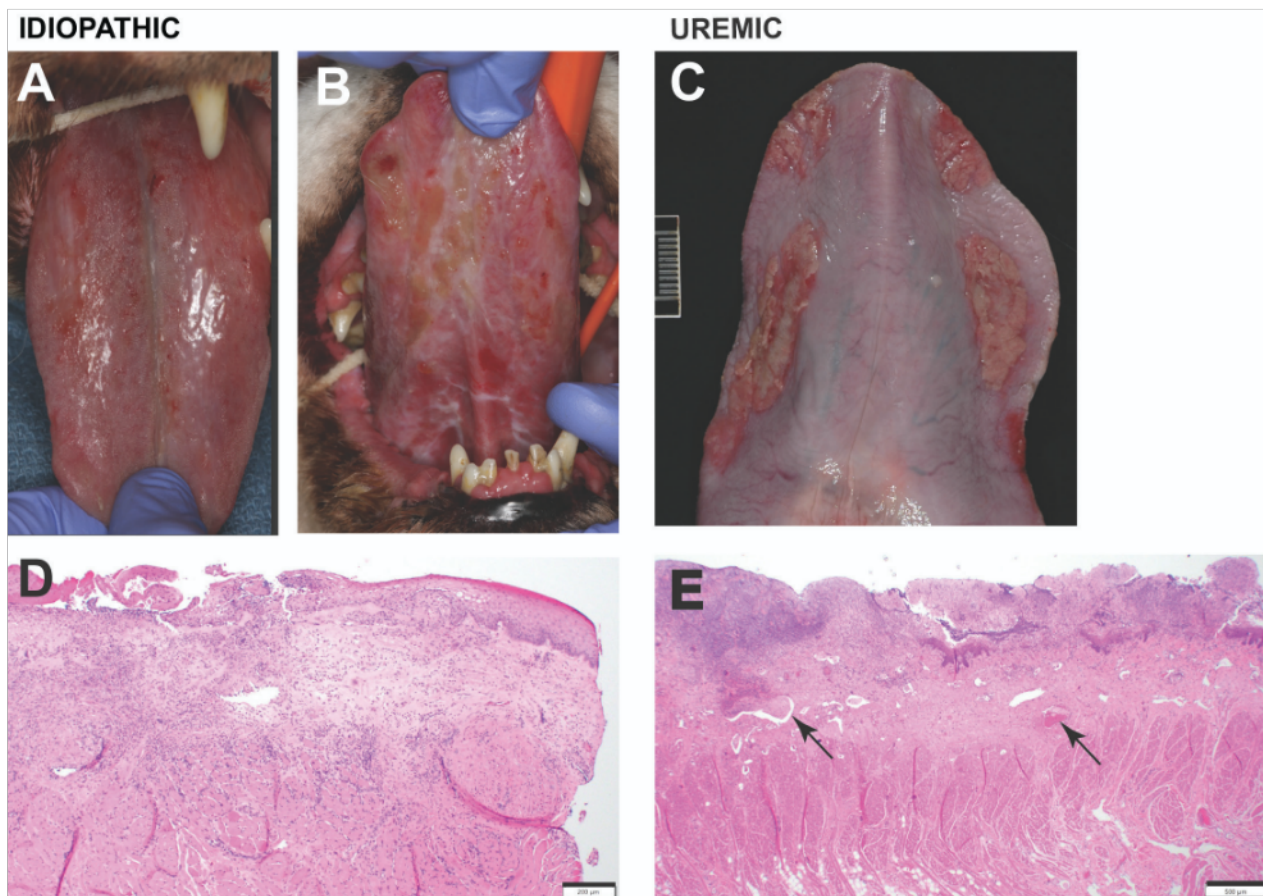


Figure 2—Clinical appearance of idiopathic glossitis presented as multifocal lesions on the dorsal surface (A) and ventral surface of the tongue (B). C—Clinical appearance of uremia-associated glossitis with bilateral involvement on the ventral surface of the tongue. D—Histopathological presentation of the lesions shown in A and B. Note the epithelial disruption and ulceration subtended by edema, myodegeneration, and inflammation. Bar = 200 μ m; H&E stain. E—Histopathological presentation of the lesion shown in panel C. Note the multifocal ulceration of the epithelium associated with inflammatory exudate. The black arrows indicate thrombosed small-caliber vessels in the lamina propria. Bar = 200 μ m; H&E stain.

mostly related to renal disease resulting in uremia-associated lesions (8 of 13 [61.5%]). Other identifiable medical causes included nonimmune-mediated vasculitis (2 of 13 [15.4%]), mucopolysaccharidosis (1 of 13 [7.7%]), systemic parvovirus infection (1 of 13 [7.7%]), and caval syndrome (heartworm disease due to *Dirofilaria immitis*; 1 of 13 [7.7%]).

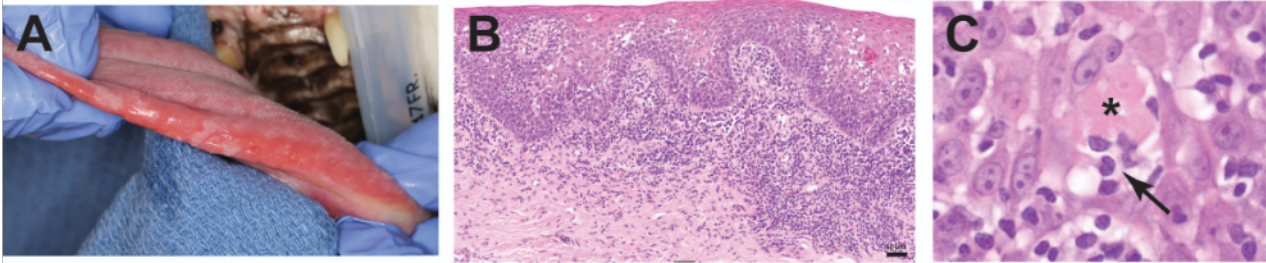
Uremia-associated glossitis was most frequently reported as bilateral, multifocal, or solitary, affecting the ventral surface of the tongue and more commonly seen at the rostral third. Histologically, glossitis of this type was mostly identified as acutely, moderately inflamed, with mostly neutrophilic inflammation, and often grossly described as ulcerative or erosive. Necrosis, intralesional bacteria, or both were detected in some lesions. In addition, blood work of 5 of the 8 dogs in our study with uremia-associated glossitis was available for review and showed that their BUN level ranged from 129 to 272 mg/dL (reference range, 5 to 33 mg/dL).

Glossitis of immune-mediated or autoimmune etiology (8 of 100 [8%]) was mostly diagnosed as erythema multiforme (5 of 8 [62.5%]). Other condi-

tions identified in this category included systemic lupus erythematosus (1 of 8 [12.5%]), “sweet syndrome-like condition” (ie, neutrophilic dermatosis; 1 of 8 [12.5%]), and immune-mediated vasculitis (1 of 8 [12.5%]). This type of glossitis was mostly reported as bilateral, affecting the lateral surface of the tongue. It was primarily grossly described as ulcerative and erosive, with lymphoplasmacytic inflammation more commonly seen histologically. Necrosis, mineralization, or both were noted in some lesions.

The diagnosis of glossitis associated with a foreign body (5 of 10 [5%]) was reached when a foreign object was identified in the lesion or presence of a migrating foreign object at a draining tract associated with the lesion. This type of glossitis lesion was mostly solitary, affecting the ventral surface of the tongue and more often located in the middle third. Histologically, glossitis of this type was identified as severely inflamed, with lymphoplasmacytic, neutrophilic, and/or histiocytic infiltration. It was also described grossly as ulcerative, erosive, or suppurative. Necrosis was detected in some lesions.

IMMUNE-MEDIATED



MUCOPOLYSACCHARIDOSIS

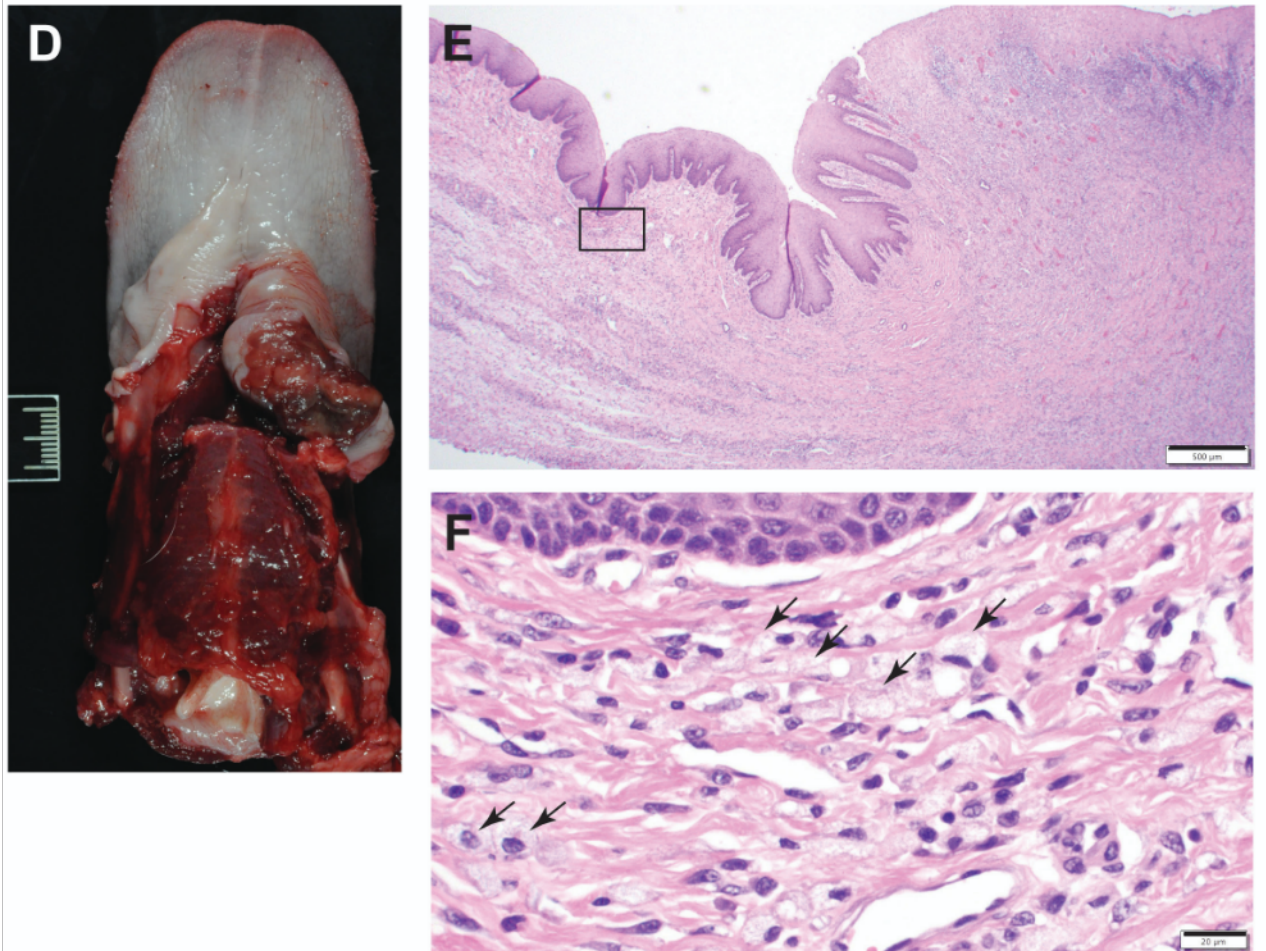


Figure 3—A—Clinical appearance of glossitis of immune-mediated etiology with the lesion seen at the lateral border of the tongue. B—Histological presentation of the lesion shown in panel A. Note the acanthosis in the epithelial layer and continuous lymphoplasmacytic infiltrate in the superficial lamina propria. Bar = 50 μ m; H&E stain. C—High magnification of the epithelium from the section shown in panel B. Note the individual cell necrosis (asterisk) accompanied by lymphocytes (arrow) (lymphocytic satellitosis), a hallmark of the erythema multiforme diagnosis with histology. D—Clinical appearance of glossitis associated with mucopolysaccharidosis, presented as an ulcerative nodular mass at the tongue base. E—Histological presentation of the lesion shown in panel D. Note the partially ulcerated and necrotic epithelium on the right aspect of the tissue section subtended by inflammatory infiltrate, edema, and additional tissue necrosis. Bar = 500 μ m; H&E stain. F—High-magnification view of the region enclosed in the black rectangle in panel E. Note the multiple foamy cells (macrophages; arrows) scattered through the interstitium, with no evidence of associated inflammation. Bar = 20 μ m; H&E stain.

Treatment and follow-up response

Treatment for (but not specific to) glossitis, concurrent medical conditions, or both was delivered in 81 dogs (81%). Types of treatment reported were medical therapy, including the use of an analgesic, an-

tibiotic, anti-inflammatory, immunosuppressant, immunomodulatory, or gastroprotectant medication (n = 68); periodontal treatment (ie, scaling and polishing; 39); surgical excision or exploration of the glossitis lesion (9); and IV fluid therapy (12). Among those, 28

INFECTIOUS (Canine Parvovirus)

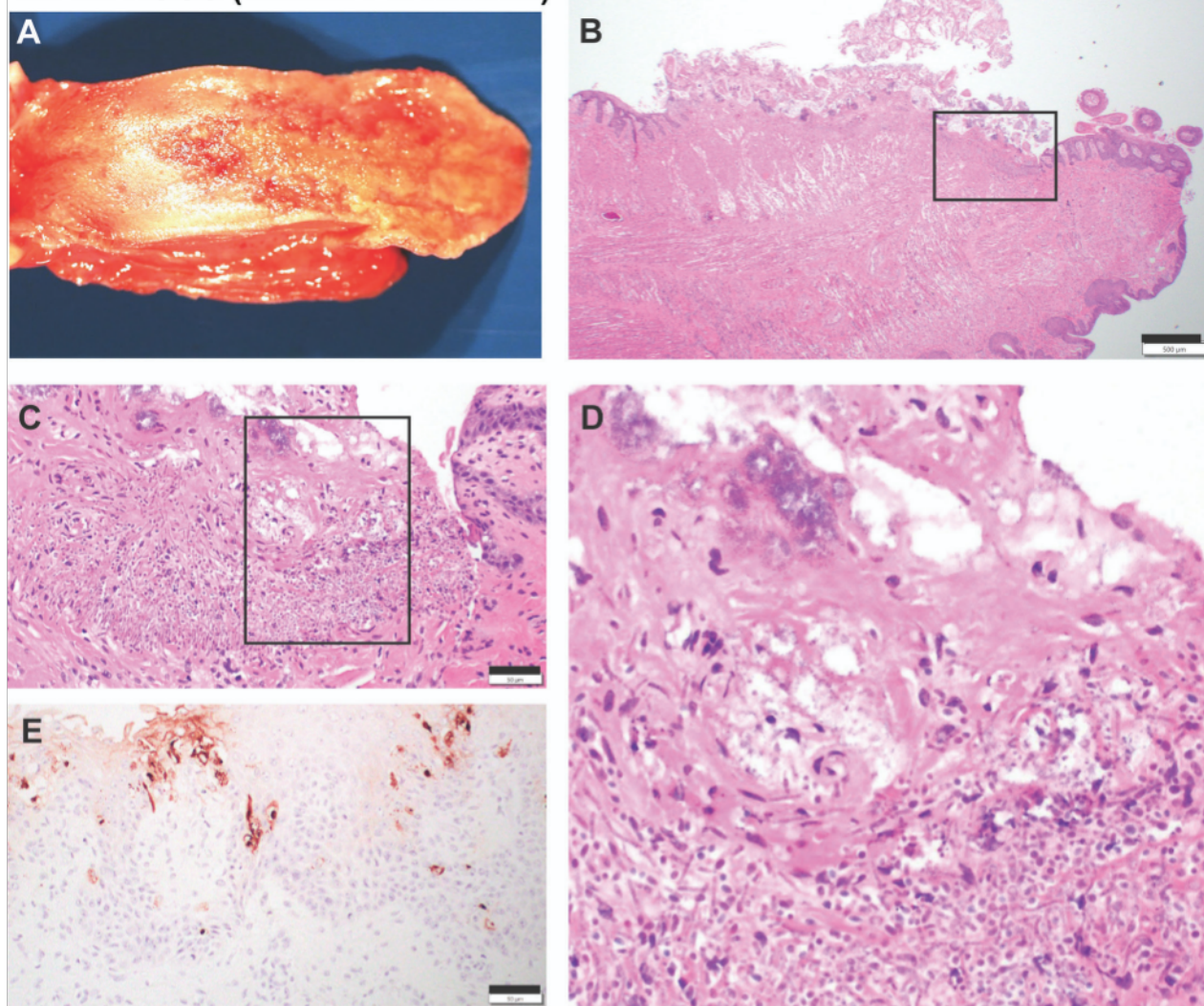


Figure 4—A—Clinical appearance of glossitis associated with systemic parvovirus infection; note that the dorsal aspect of the tongue is multifocally eroded. B—Low-magnification histological presentation of the lesion displayed in panel A. Note the segmental epithelial ulceration overlain by eosinophilic amorphous material (cellular debris, fibrin, and inflammatory cells). Bar = 500 μ m; H&E stain. C—Higher-magnification image of the area enclosed in the black rectangle in panel B. Note the superficial layer of fibrin admixed with inflammatory cells subtended by a mat of fungal elements. Bar = 10 μ m; H&E stain. D—Very high magnification of the area enclosed in the black rectangle in panel C. Note the round yeasts and short hyphal structures as well as the bacterial colonies in the exudate. E—Immunohistochemical labeling for canine parvovirus. Note the strong immunoreactivity in the epithelial cells of the tongue. Bar = 10 μ m; canine parvovirus immunohistochemistry.

dogs (34.6%) were managed by 1 type of treatment, whereas 51 dogs (62.9%) and 2 dogs (2.5%) received 2 and 3 types of treatment, respectively.

Follow-up data on glossitis lesions were available in 43 cases (43%). Median (IQR) follow-up time was 21 days (14 to 28 days). Most dogs (38 of 43 [88.4%]) had 1 follow-up appointment. Three dogs (7.0%) had 2 follow-up appointments. The remaining 2 dogs attended 3 and 5 rechecks (2.3% each). Glossitis was deemed to be healed at the last recheck appointment in 22 cases (51.2%), healing with improvement seen in 18 cases (41.8%), and persistent without improvement or recurrence of the lesion in 3 cases (7.0%).

Discussion

This retrospective study characterized the presentation, clinical features, and histopathological findings of glossitis in dogs and determined the clinical-pathological correlation of this disease entity. Several clinically important findings were described. First, awake oral examination failed to detect 55% of lingual lesions that were later confirmed as glossitis on histopathology. Second, 6 inciting causes of glossitis in dogs were identified, with trauma and idiopathy being the most common causes, followed by inflammatory (associated with concurrent local oral or dental disease), oral manifestation of systemic disease (other than immune-

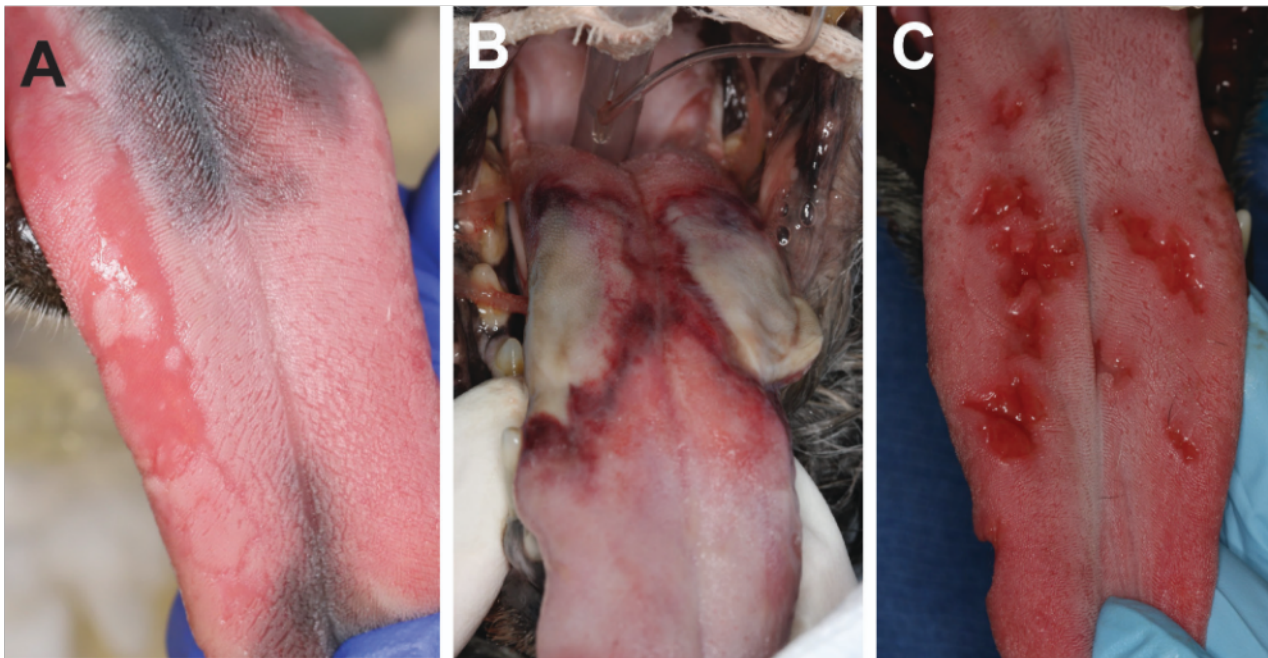


Figure 5—A—Clinical presentation of glossitis of inflammatory cause; note the unilateral lesion affecting the dorsal surface and lateral border of the tongue. B—Clinical presentation of idiopathic glossitis presented as extensive bilateral necrotic lesions. C—Clinical presentation of glossitis that resulted from caustic injury, in which the lesions are multifocally distributed on the dorsal surface of the tongue.

mediated or autoimmune), immune-mediated or autoimmune, and related to a foreign body. Third, the appearance and presentation of different causes of glossitis varied; glossitis of a specific cause appeared to have specific clinical and histological patterns. Glossitis commonly presented as either a unilateral or bilateral lesion, affecting the ventral surface of the middle or rostral third of the tongue; the predominant histological features were chronic, severe ulcerative or erosive neutrophilic inflammation.

We demonstrated that only 19% of dogs diagnosed with glossitis had an initial presenting complaint related to the tongue, and almost 40% of dogs had signs of dysphagia upon further history-taking. The data signified the limitation of the awake oral examination and further revealed the possibility of glossitis going unnoticed. The middle and caudal thirds of the tongue are relatively difficult to assess because of their inherent relatively immobile attachment to other oral structures and they are typically spontaneously retracted during awake oral examination. Anesthetized oral examination allows for a thorough visual and tactile assessment of oral soft tissues including the tongue and determines the need for and choice of further diagnostics.

Interestingly, several clinical records in our study stated that the lingual lesion was detected by the anesthetist at the time of intubation for general anesthesia for diagnostics unrelated to the lingual lesion. Considering that the cases in our study were evaluated by various specialty services and disciplines, adoption of a multidisciplinary approach in detecting and managing this disease entity appears to be beneficial to the patient. Furthermore, advanced imaging, although uncommonly used in our cohort, was mainly

used in cases associated with trauma or foreign body, with CT scan (with contrast) being the most chosen modality. In fact, advanced imaging allows evaluation of the extent of the lingual lesion and its deep layers,¹² and possibly helps with foreign body detection depending on the material type,^{13–15} and its retrieval from the tongue.¹⁶ Moreover, a recent case report¹⁷ demonstrated the use of thermographic imaging to track the lingual perfusion in a dog with macroglossia caused by strangulating linear foreign body. We therefore advise the rational use of advanced imaging for glossitis, as it complements the diagnostic process and aids in treatment planning, especially in cases with suspect foreign body entrapment.

Previous studies of lingual lesions conducted by means of reviewing histopathology reports alone stated that the inciting causes of most glossitis cases in dogs are inapparent and the definitive cause of glossitis was usually not evident histologically.^{3,4} However, we demonstrated that correlating data from patient history, clinical examination findings, radiological findings (if any), and histopathological results can aid in the identification of specific etiology. Nevertheless, 30% of the glossitis cases had inciting causes that remain unknown.

Proposed causes of glossitis discussed in the veterinary literature are trauma, bite wounds, (migrating) foreign bodies, electric burns, caustic agents, infectious causes (eg, canine calicivirus, canine distemper virus, *Pasteurella multocida*, *Candida* spp, and various opportunistic bacteria), idiopathy, immune-mediated disease, autoimmune disease, and uremia.^{5–8} Similarly, our study identified the 2 most common causes of glossitis as traumatic and idiopathic. We detected uremia-related and im-

mune-mediated or autoimmune disease as causes of glossitis to be infrequent, whereas foreign body and infectious causes were rare. In humans, potential causes of glossitis are anemia (especially iron-deficiency anemia), vitamin deficiency, infection (viral, bacterial, fungal, and parasitic), medication-induced, exposure to irritants, mechanical trauma, poor hydration, and autoimmune conditions, among others.⁸ Anemia, vitamin deficiency, and fungal or parasitic infection were not identified as the cause of glossitis in our dog population, suggesting that there are likely differences in disease entities and pathophysiology between species. Hence, the clinical patterns of glossitis in human medicine should not be applied to canine patients.

The number of glossitis cases caused by trauma or foreign body might be underreported in the present study, especially in situations where a diagnosis could be made by means of history and clinical examination. For instance, when a specific traumatic event is witnessed or the clinical appearance of the lingual lesion matches the insult described in the history, clinicians may not opt for histopathology to confirm the diagnosis of glossitis. Besides, other mucosal lesions that occur simultaneously with glossitis in inflammatory disease (ie, canine chronic ulcerative stomatitis) and immune-mediated or autoimmune disease (ie, erythema multiforme and systemic lupus erythematosus) with more dramatic cutaneous manifestation may be prioritized over the tongue for histopathological sampling and assessment.

Notably, we reported 2 specific medical conditions that correlated with glossitis in our cohort, namely mucopolysaccharidosis (unknown type) and canine parvovirus (CPV) infection. Mucopolysaccharidosis is a rare inherited metabolic disorder that occurs in humans, dogs, and cats. The disease develops because of a deficiency of the lysosomal enzyme used for degrading glycosaminoglycans, resulting in the accumulation of glycosaminoglycans in the brain and other tissues and leading to cellular degeneration, increased infiltration of histiocytes, and multiorgan dysfunction.¹⁸ The common clinical signs include growth retardation, skeletal deformities, corneal cloudiness, facial dysmorphism, and neurological signs. The oral manifestation of this condition in humans is an enlarged tongue (macroglossia).¹⁹ In the present study, the 1-year-old spayed female Boston Terrier with this disease was initially presented for ataxia, general weakness, and dysphagia. The final diagnosis of mucopolysaccharidosis was obtained by necropsy. The glossitis was grossly described as a 10 X 15 X 25-mm, raised, ulcerative, nodular mass that exuded purulent material on the left tongue base, with histopathology assessment as moderate, focally extensive, proliferative, ulcerative, histiocytic, and neutrophilic glossitis. Special staining (Alcian blue and periodic acid-Schiff staining) was applied to the sample, revealing a strong positive staining of vacuolated "clear cells." Although macroglossia was not specified in our record, to our knowledge, this documentation was the first of mucopolysaccharidosis as an inciting cause of glossitis in a dog.

Canine parvovirus infection is caused by CPV type 2 (CPV-2), which results in parvoenteritis and generalized lymphocyte depletion.²⁰ Canine parvoenteritis, with the virus affecting gastrointestinal tissues including the epithelium of the tongue, oral cavity, esophagus, and intestinal tract, can result in severe morbidities and mortality.²¹ Ulcerative glossitis may occur in some puppies,²¹ matching with our finding of a 2-month-old female Dachshund infected with CPV-2 that had a histologically diagnosed severe, multifocal, ulcerative glossitis on the dorsal aspect of the tongue, with presence of intralesional yeast (consistent with *Candida* spp) and bacterial cocci colonization.

Results of the present study indicated that the clinical appearance and presentation of different causes of glossitis vary. Although the number of cases of each inciting cause was limited, glossitis of a specific cause appeared to trend toward a specific clinical and histological pattern. Our data on glossitis related to self-induced trauma (chewing) were mostly aligned with a recent report of oral fibroepithelial polyps (chewing granuloma),²² except that our cases exhibited neutrophilic inflammation more commonly than lymphoplasmacytic inflammation, indicating a possible acute component on the chronic condition. In addition, our study demonstrated that glossitis of immune-mediated or autoimmune etiology is bilaterally involved, affecting the lateral surface of the tongue more commonly. Histologically, lymphoplasmacytic inflammation dominated, fitting with the pathogenesis of immune-mediated or autoimmune disease. Furthermore, our findings on uremia-associated glossitis matched with a previous description,⁷ in which it is typically multifocal and necroulcerative and occurs along the ventral aspect of the tongue; buccal lesions are observed. The BUN level of the 5 dogs with uremia-associated glossitis ranged from 129 to 272 mg/dL, whereas, in humans, uremic stomatitis occurs in end-stage renal failure with a reported BUN level higher than 150 mg/dL.²³ While the threshold of BUN that correlates with the development of stomatitis or glossitis in dogs has not been established, our findings lay the basis for future study on the relationship between BUN values and uremia-associated oral mucosal lesions.

It is important to note that epithelial dysplasia was detected in 5 glossitis cases of this study. Two lesions were associated with trauma, and 3 were idiopathic. Histologically, epithelial dysplasia can be an individual feature, but if extensive, it can be a diagnosis itself and become a concern because of the potential for malignant transformation.²⁴ In human medicine, oral epithelial dysplasia is a potentially premalignant disorder of the oral mucosa, with a risk of malignant transformation to squamous cell carcinoma. Although there are mixed opinions about the association between chronic mucosal trauma or inflammation and oral squamous cell carcinoma in humans,²⁵⁻²⁷ no such relationship has been established in cats²⁸ and has not been studied in dogs. Nevertheless, our data emphasize the need for biopsy of oral lesions for histopathology analysis. It allows for evaluation at both the tissue and cell levels to determine the disease process and correlate such findings

with clinical data to reach a diagnosis and aid with treatment planning.

In conclusion, this retrospective study characterized the presentation, clinical features, and histopathological findings of glossitis in dogs and determined the clinical-pathological correlation. Glossitis may be presented as a primary disease or manifestation of underlying medical conditions, highlighting the importance of obtaining history, completing a thorough clinical examination, and using proper diagnostics in a multidisciplinary approach, including histopathology assessment as a gold standard for definitive diagnosis and treatment planning.

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Supplementary Materials

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