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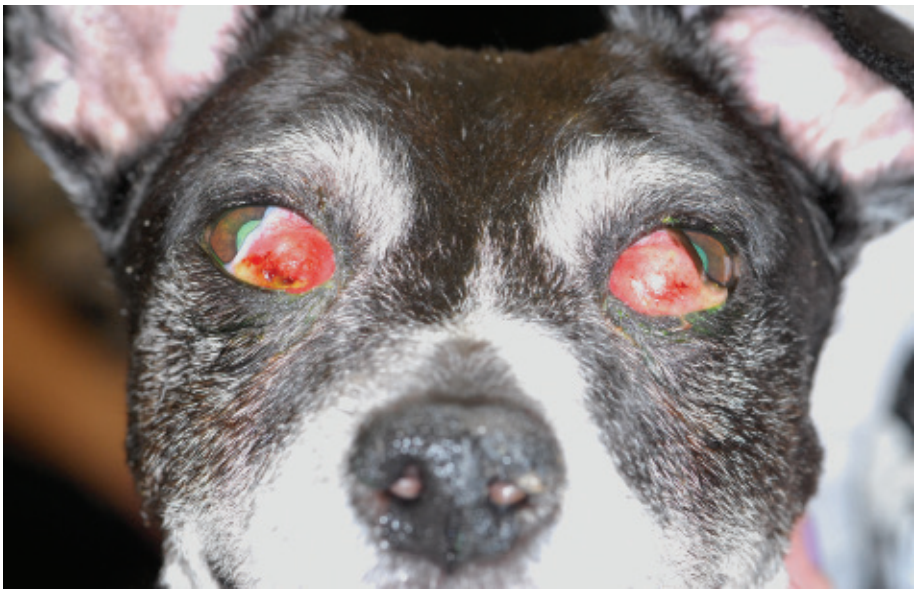

Figure 1—Photograph of a 9-year-old Terrier mix dog that was evaluated because of so-called red eyes of 2 days' duration. Notice the bilateral third eyelid prolapse with severe hyperemia as a result of exposure.

History

A 9-year-old 14.6-kg (32.12-lb; body condition score, 4/5) spayed female Terrier mix dog was referred for evaluation of bilateral third eyelid prolapse of 2 days' duration. No history of previous ocular disease was reported. The owner had noticed that the dog had had polyuria and polydipsia for several months.

Clinical and Gross Findings

Ophthalmic examination findings for the dog included a positive maze test response and positive menace, palpebral, oculocephalic, dazzle, and pupillary light reflexes bilaterally. Schirmer tear test results were 17 mm for the right eye and 14 mm for the left eye. For the eyes of healthy dogs, tear test findings should exceed 15 mm in 1 minute.[1] Intraocular pressure was 22 mm Hg in the right eye and 30 mm Hg in the left eye. The reference range for intraocular pressure in dogs is 8 to 25 mm Hg.[1] Retropulsion was not possible in either eye. The result of a nasolacrimal test was negative for both eyes. Fluorescein stain uptake was present unilaterally (right eye). The third eyelid of each eye was prolapsed with severe hyperemia and suspected ulceration **(Figure 1)**. Signs of pain were elicited during attempts to open the dog's mouth.

Ocular ultrasonography revealed a 2-cm-diameter, irregularly shaped, well-defined hypoechoic structure caudal to each globe. The bilateral masses appeared adjacent to and separate from the optic nerve. Ultrasonographically, the globes were unremarkable. Further cross-sectional imaging was recommended, but the owner declined such procedures.

A CBC, serum biochemical panel, and urinalysis were performed. The CBC results were unremarkable. The serum biochemical panel and urinalysis findings were consistent with diabetic ketoacidosis as well as renal and hepatic damage attributable to diabetes mellitus. Owing to the dog's poor prognosis and quality of life, euthanasia by IV injection of pentobarbital was performed.

During the postmortem examination of the dog, notable gross findings were confined to the ocular system. The third eyelid of each eye was prolapsed, reddened, and swollen. No masses were evident in the retrobulbar space, but the retrobulbar tissues appeared unusually soft.

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

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Histopathologic Findings

The outer surfaces of the third eyelids were ulcerated and covered by karyorrhectic cellular debris mixed with fibrinous exudate and numerous neutrophils. The periocular fat was partially effaced by similar exudate mixed with some macrophages, lymphocytes, and plasma cells. The grossly soft retrobulbar tissues included an extensively necrotic and mildly inflamed zygomatic salivary gland with lakes of free mucin and multifocal fibrin deposits **(Figure 2)**. The adjacent musculature had features of myocyte degeneration and early regeneration. Areas of early fibrosis dissected the intervening adipose tissue. Alterations in the globes were restricted to a delicate pre-iridal fibrovascular membrane in both eyes. In sections of the zygomatic salivary glands, no infectious agent was detected with special (Giemsa and Gram) stains.

The dog also had moderate pancreatic islet cellular vacuolation, mild hepatocellular fatty degeneration, and mild renal glomerulopathy. These findings were consistent with the antemortem diagnosis of diabetes mellitus.

Morphologic Diagnoses and Case Summary

Morphologic diagnosis: bilateral, acute to subacute, necrosuppurative zygomatic sialadenitis and periocular steatitis with bilateral prolapse of the third eyelid and acute, necrosuppurative conjunctivitis.

Case summary: zygomatic sialadenitis causing bilateral third eyelid prolapse in a dog.

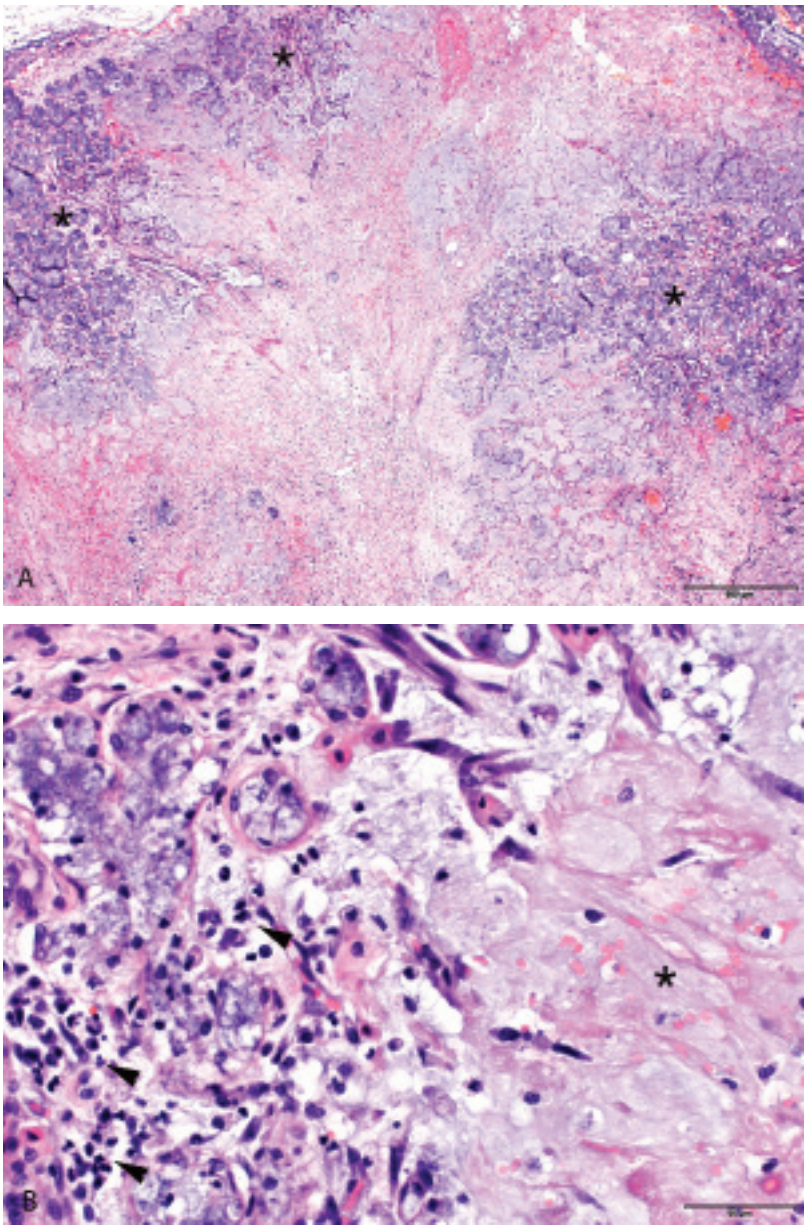

Figure 2—Photomicrographs of sections of 1 of the dog's 2 zygomatic salivary glands. A—The gland is extensively necrotic with complete loss of the mucous secretory units and their replacement by lakes of mucin, fibrin, and hemorrhage. The normal glandular tissue (asterisks) remains viable in a few areas only. H&E stain; bar = 500 µm. B—At higher magnification, the parenchyma of the mucous gland is replaced by pale amphophilic mucin, eosinophilic strands of fibrin, and extravasated erythrocytes (asterisk). Inflammatory cells, predominantly neutrophils (arrowheads), have infiltrated both the necrotic and remaining viable peripheral glandular tissue. H&E stain; bar = 50 µm.

Comments

For the dog of the present report, an initial ultrasonographic diagnosis of retrobulbar neoplasia, abscess, or granuloma was made; the possible tissues of origin included salivary gland, extraocular muscles, or other ocular adnexa. Dogs have 4 paired major salivary glands: parotid, sublingual, mandibular, and zygomatic. Each zygomatic salivary gland is ventromedial to the zygomatic arch and pyramidal in shape, with its base located dorsocaudal and adjacent to the ventral part of the periorbital area. The apex of the gland is lateral to the most caudal maxillary tooth and sends 1 major and 2 to 4 minor ducts to an area immediately caudal to the parotid papilla.[2]

Sialadenitis is defined as inflammation of a salivary gland, with the zygomatic glands being least often affected.[3] The reported incidence of salivary gland disease in dogs is < 0.3%, and 26% of those cases involve sialadenitis.[3] Because of its rare occurrence, zygomatic sialadenitis is often overlooked. However, zygomatic gland disease should be on the differential diagnosis list for any dog with periorbital swelling, protrusion of the third eyelid, and any other clinical signs of retrobulbar disease.

In terms of predisposition to zygomatic sialadenitis among dogs, the veterinary medical literature

is somewhat contradictory. Some sources report no predilection with regard to age, breed, or sex, whereas others suspect increased prevalence in certain breeds such as the Beagle.[3] In 1 study,[4] most affected animals were middle-aged (mean age, 7.7 years) medium- to large-breed dogs that had unilateral disease. There are some reports[3–5] of greater frequency of the disease in male dogs. The definitive etiopathogenesis of zygomatic sialadenitis remains unknown; however, immune-mediated disease, trauma (foreign body), expanding regional inflammation, and infection (systemic [eg, canine distemper virus] or localized) have all been proposed as possible causes.[3,4,6] Sialadenitis has also been linked to afferent vagal impulses and limbic epilepsy in some dogs; this subset of dogs responds to treatment with phenobarbital.[6–8]

The clinical signs most suggestive of zygomatic gland disease are zygomatic papilla swelling and hypersalivation.[4,6,7] Much more often, zygomatic gland disease results in signs of retrobulbar disease because of the gland's proximity to the eye.[4,5] In a retrospective study[4] that evaluated the clinical and diagnostic imaging findings for 11 dogs with zygomatic sialadenitis, various signs of retrobulbar disease were evident, including protrusion of the third eyelid, exophthalmos, decreased or absent retropulsion, and signs of pain on opening the mouth. Additional nonspecific signs associated with zygomatic salivary gland disease include conjunctival and episcleral hyperemia, chemosis, ocular discharge, and divergent strabismus.[4–6,a] Some dogs also develop blindness.[4,6]

Advanced imaging of the orbit, often as part of evaluation of the head, and histologic examination of samples from the affected gland and other orbital tissues are usually needed to confirm salivary gland involvement and to determine the underlying cause of disease.[4,5,a] Radiography is a procedure of low diagnostic usefulness in these cases.[a] Ocular ultrasonography provides limited evidence of zygomatic gland disease; however, it is a useful aid to guide fine-needle aspiration of abnormal tissues.[4] Although more expensive, MRI and CT offer the most accurate imaging of the orbit and provide the most useful information for surgical planning.[4,5,a]

In dogs with zygomatic sialadenitis, results of clinicopathologic analyses are typically within reference ranges, but neutrophilia and monocytosis secondary to associated inflammation may develop.[4,5] Cytologic examination of ultrasound-guided tissue aspirate preparations usually reveals inflammatory cells, epithelial cells, and mucin.[4,5] A sialocele is almost always present, and it is difficult to determine whether its development precedes or follows the onset of sialadenitis.[3–6] Microbial culture of aspirated tissue specimens usually yields no growth, although small numbers of bacteria were detected in specimens from a few dogs in 1 report.[4] Histologically, lobular epithelial degeneration and necrosis are often present.[3,4,6]

In addition to sialadenitis and sialoceles, another important cause of salivary gland enlargement is salivary gland neoplasia. In a large retrospective study[3] of salivary gland diseases in dogs and cats, 41 of the 160 canine salivary gland specimens were neoplastic. In general, salivary gland neoplasms are primary tumors (largely adenomas, carcinomas, and mixed tumors) but can develop secondary to other neoplasms, such as lymphoma.[3]

Treatment of sialadenitis includes oral administration of antimicrobial and anti-inflammatory drugs, as well as supportive care for the signs associated with orbital cellulitis.[4,6,7] Neomycin-polymyxin-bacitracin or petrolatum-based ocular ointments may be used to treat the ophthalmic signs.[4] Various combinations of systemically administered antimicrobials and anti-inflammatory drugs (eg, amoxicillin–clavulanic acid, enrofloxacin, prednisone, carprofen, and deracoxib) have been successful treatments.[4] Some affected dogs also respond to phenobarbital.[6–8] If sialadenitis is refractory to medical management, a modified lateral orbitotomy with zygomatic arch osteotomy and salivary gland removal, along with temporary tarsorrhaphy or drainage tube placement, may be performed.[4,5,a]

Relapse of sialadenitis after medications are discontinued may occur, although most dogs can be weaned off treatments slowly and avoid disease recurrence.[4,6,7] Depending on the underlying cause of sialadenitis, flare-ups may occur throughout an affected dog's life. However, despite this possibility of recurrence, the prognosis for dogs with sialadenitis is generally good.

Footnotes

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