Sialadenosis in dogs

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Objective—To determine clinical findings, cytologic and histologic characteristics of salivary glands, and response to treatment with phenobarbital in dogs with clinical signs typical of sialadenosis.

Design—Prospective study.

Animals—13 dogs with enlarged salivary glands.

Procedure—Data were collected from dogs with clinical signs attributable to enlarged salivary glands. Salivary gland biopsy and cytologic specimens were examined. Dogs were treated with phenobarbital and monitored for response to treatment.

Results—Clinical signs commonly associated with sialadenosis included retching and gulping. Substantial cellular changes were not detected by histologic or cytologic examination of enlarged salivary glands. Response to treatment with phenobarbital was rapid, although most dogs required continuous treatment to prevent recurrence of clinical signs.

Conclusions and Clinical Relevance—Sialadenosis is a condition of unknown cause that may have been underdiagnosed in dogs. Criteria for diagnosis include typical clinical signs, enlarged salivary glands, and lack of substantial microscopic lesions. Response to treatment with phenobarbital is rapid. 

Sialadenosis in humans is described as a bilateral, uniformal, painless, and noninflammatory enlargement of the salivary glands; the parotid salivary glands are more commonly affected than the mandibular salivary glands. Sialadenosis comprises 6% of diagnoses of salivary gland disease in humans and may develop as a result of physiologic hypertrophy in patients with compulsive eating disorders or in association with acromegaly, but in many instances the cause remains obscure. Vomiting is a common clinical sign and, in bulimic patients, the frequency of vomiting increases in proportion to the degree of salivary gland enlargement. Sialadenosis has been recognized after administration of a variety of drugs and in association with endocrine disorders such as diabetes. A primary abnormality of the sympathetic innervation to the salivary glands has been proposed as a possible cause and may be associated with certain eating disorders, such as bulimia.

Although a series of 245 cases of salivary gland disease did not include sialadenosis, another report described findings consistent with this diagnosis in 2 dogs that responded well to treatment with phenobarbital. The objective of the study reported here was to determine clinical findings, cytologic and histologic characteristics of salivary glands, and response to treatment with phenobarbital in dogs with clinical signs typical of sialadenosis.

Materials and Methods

Dogs—During a 7-year period (1990 to 1997), 13 dogs were enrolled in the study on the basis of history and clinical findings that included excessive salivation, poor appetite, and submandibular swelling or exophthalmos caused by bilateral, nonpainful enlargement of the mandibular or zygomatic salivary glands.

Clinical examinations—History and results of clinical examinations, including physical examination, CBC, serum biochemical analyses, thoracic radiography, esophageal endoscopy, and ultrasonography, were recorded. Unilateral excision of 1 mandibular gland was performed on 8 dogs to obtain specimens for histologic examination, fine-needle aspiration was performed on 6 dogs, and 1 dog received both procedures. Histologic and cytologic examinations were performed at several commercial and university laboratories.

Treatment—For 12 of the 13 dogs, treatment with phenobarbital (1 mg/kg [0.45 mg/lb] of body weight, PO, q 12 h) was initiated 2 weeks after biopsy or fine-needle aspiration was performed, when it was determined that improvement in clinical signs had not developed. One dog was examined for keratoconjunctivitis sicca and salivary gland enlargement that developed 3 weeks after vaccination against canine distemper virus, parvovirus, adenovirus, parainfluenza virus, and Leptospira spp; an immune-mediated disorder was suspected, and treatment was initiated with prednisolone (2 mg/kg [0.91 mg/lb], PO, q 12 h). Clinical response was not detected after 1 week, so treatment with azathioprine (1 mg/kg [0.45 mg/lb]) and topical administration of cyclosporine and dexamethasone was instituted. Clinical signs of salivary gland enlargement and retching continued unabated; administration of phenobarbital was initiated 4 weeks after initial evaluation. Dogs were reexamined 1 and 4 weeks after initiation of treatment with phenobarbital, and telephone follow-up was maintained (range, 8 to 48 months).

Results

Historical and clinical findings—Breeds included English Springer Spaniel (n = 2), Border Collie (2), Fox Terrier (Wire [1]), Jack Russell Terrier (1), Beagle (1), Cavalier King Charles Spaniel (1), Yorkshire Terrier (1), West Highland White Terrier (1), English Bulldog (1), and mixed breed (2). Age of dogs ranged from 1 to 17 years (median, 4.5 years); 1 dog was a sexually intact male, 5 were castrated males, 1 was a sexually intact female, and 6 were spayed females. For all dogs, clinical signs were reported to have had sudden onset and had been noticed for at least 2 months (range, 2 to 18 months). Typically, dogs were examined for episodes of retching or gulping that were elicited by mild excitement and occurred several times each day. Other reported clinical signs were weight loss, reluctance to exercise, snorting, lip smacking, nasal discharge, drooling, reduced food consumption, and signs.
of depression. For all dogs except 1, prior treatments had included a variety of drugs, including antimicrobials, corticosteroids, and parasympathomimetic agents.

Results of physical examinations were unremarkable except for bilateral mandibular salivary gland enlargement to approximately twice normal size (n = 11) and exophthalmos (2). Dogs with submandibular swellings were particularly sensitive to gentle external palpation of the throat, compared with clinically normal dogs; palpation did not elicit signs of pain but caused retching and gulping. Results of hematologic and serum biochemical analyses and thoracic radiography did not reveal abnormalities. Esophageal endoscopy did not reveal abnormalities in 12 dogs. In 1 dog, reduced esophageal muscle tone was attributed to effects of anesthesia. Esophageal function as assessed by barium swallow was not performed. Ultrasonography was used to assess size of the mandibular salivary glands and detected a homogeneous mass ventrolateral to the posterior aspect of the right globe of 2 dogs with exophthalmos, which was interpreted to represent the zygomatic salivary gland. Cytologic and histologic examinations revealed normal salivary gland tissue in 9 dogs and mild salivary epithelial hypertrophy in 4 dogs.

All owners reported substantial improvement in clinical signs (retching, gulping) in the 11 dogs with mandibular salivary gland enlargement within 24 to 36 hours of commencing oral administration of phenobarbital, and clinical signs had resolved within 1 week, although no changes had been noticed after excision of a salivary gland. Subjectively, enlargement of the mandibular salivary glands was reduced in all dogs after 2 weeks of treatment. Improvement continued for 10 dogs; for 1 dog, owner compliance was poor, and the dog was euthanatized at the owner’s request. Treatment was continued in the remaining dogs, because owners reported recurrence of clinical signs when treatment was discontinued. Exophthalmos in 2 dogs had completely resolved after 4 months of treatment. The treatment was discontinued, and recurrence of clinical signs has not developed.

Discussion

To the authors’ knowledge, sialadenosis has not been described in dogs, although there are several reports of similar conditions. In the dogs reported here, clinical signs were not accompanied by endocrine disorders or other autonomic neurologic deficits that have been reported with sialadenosis in humans. Reching and gulping may have been caused by excessive saliva production, but this could not be confirmed. Excessive saliva production may be associated with increased parasympathetic activity; changes in sympathetic innervation also have been proposed. Innervation of the salivary glands of the dogs reported here was not investigated. The fact that clinical response to excision of 1 salivary gland was not detected suggests that the physical effect of gland enlargement may not have been an important cause of clinical signs such as retching.

The use of inspection and palpation to determine the size of salivary glands in humans has been considered suitable for diagnosis. Gland enlargement caused by hyperplasia or hypertrophy is difficult to confirm histologically because of the variability in acinar appearance caused by accumulation of secretions or contracture. Increase in size of individual acinar cells has been described, but morphometric studies performed recently in dogs have not confirmed this finding. Imaging techniques that may be used to detect salivary gland swelling include sialography, ultrasonography, and computed tomography, but results may not be definitive, and diagnosis requires correlation with clinical findings and patient history. Examination of histologic or cytologic specimens is important to confirm the lack of microscopic lesions that is characteristic of the disease.

Response to oral administration of phenobarbital was rapid, considering the time required to reach plasma concentrations that are therapeutic for the control of seizure activity (10 to 14 days), which may suggest that phenobarbital was effective at low concentrations. Oral administration of pilocarpine has also been used; effects were beneficial but inconsistent.

References


