Animal Behavior Case of the Month

This feature is sponsored by the American College of Veterinary Behaviorists. Readers of the *JAVMA* are invited to submit reports, which should include a brief description of a behavioral problem, the evaluation and treatment, and a succinct discussion of the case.

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Statement of the Problem

A 9-year-old Toy Poodle was evaluated because of progressive self-mutilation of the right nasal area. The dog also had a history of repetitive licking of items in its environment.

Signalment

The dog was a 7.5-kg (15-lb) neutered male Toy Poodle that had been born in December 1983.

History

The dog (dog 1) belonged to a veterinarian who permitted it to roam her clinic freely for most of its life, greeting and receiving the attention of incoming clients. The veterinarian had obtained the dog at 6 weeks of age to provide companionship for her existing dog, also a Toy Poodle. For approximately 18 months, the dogs seemingly enjoyed each other's company. When the first dog died, the owner purchased a female Toy Poodle (dog 2) as a companion for dog 1. Dog 1 learned quickly that sneezing on command in the clinic reception area and examination room generated immediate rewards in the form of attention and applause, and children in the reception area encouraged dog 1's sneezing behavior. Dog 2 soon learned to sneeze on command. Daily, the 2 dogs accompanied clients and patients to the examination room, where they sat on a bench and observed veterinary examinations performed by their owner.

At 7 years of age, dog 1 developed a behavioral disorder consisting of repetitively licking objects in its environment. The licking behavior became progressively more frequent and more intense over time. Contemporaneously, dog 2 was bred to a Yorkshire Terrier. Two of the pups from the litter were kept by the owner, thus increasing competition for various resources around the home. The new pups spent their days with the veterinarian's husband, only interacting with dogs 1 and 2 at home during evening hours. Dog 1 usually licked when it was not the focus of attention or was being ignored. For example, dog 1 would lick the seats of the car if dog 2 was sitting in the owner's lap and would lick the carpet at home at dinnertime if no attention was being paid to him.

At 9 years of age, dog 1 developed a new behavior that consisted of letting out a sharp cry and attacking the right nasal region of its face with its right hind foot. Occasionally, the dog attacked the left side of its face, rather than the right side, but this occurred only about 3 or 4 times. Attacks usually lasted 2 minutes and would usually take place in the early morning, while dog 1 was in a crate in its owner's bedroom. Initially, episodes of facial attacking occurred 3 to 4 times a month. But over a period of a few days, the attacks became more frequent, with episodes occurring several times daily. The duration of the attacks increased, sometimes lasting up to 5 minutes, as did the intensity. Touching the right side of dog 1's nose would usually initiate a bout of intense facial scratching and screaming. The dog's owner tried ignoring the behavior; however, if other people were present, they would often talk to or coddle the dog. Dog 1 was no longer permitted to roam the clinic and was confined to a cage in the clinic.

Physical Examination Findings and Laboratory Results

Results of a physical examination were unremarkable, as were results of a CBC and serum biochemical analyses. Although intradermal allergy testing was not conducted, the clinical consensus of the 4 veterinarians in the owner's practice was that allergy was not involved. They came to this conclusion on the basis of the fact that dog 1 did not have any obvious skin lesions or any evidence typical of allergies, such as foot chewing, alopecia, and pruritus. On occasion, dog 1 had mild otitis externa that responded well to topical treatment. Because scabies was endemic to the area, all 4 of the owner's dogs were treated with ivermectin for 4 weeks to rule out the possibility that sarcoptic mange was involved. No change in intensity, frequency, or duration of facial attacks resulted from this treatment. A test dose of a long-acting preparation of methyl prednisolone acetate (20 mg, IM) resulted in some improvement in dog 1's facial attacks for a period of 2 to 3 weeks, but the improvement was only modest, and the behavior was far from eliminated. Periodically, dog 1's owner would administer prednisolone acetate to provide dog 1 some respite from this seemingly stressful condition. However, dog 1 received no more than 6 injections of methyl prednisolone acetate for this purpose.

Because dog 1's dam had had tonoclonic seizures, the owner consulted a veterinary neurologist in an attempt to determine whether dog 1's problem could be, in some way, related to seizures. The neurologist, however, was of the opinion that dog 1 displayed no

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evidence of atypical seizure activity. In June of 1992, dog 1's owner consulted by telephone with the Neurology Department at the Cornell University College of Veterinary Medicine. Specialists there thought that dog 1 might have had trigeminal neuralgia and suggested a trial of treatment with carbamazepine (25 mg, PO, q 12 h, for 3 weeks). Initially there was some modest improvement, as the frequency of attacks was reduced by about 20%. However, the improvement was not sustained and lasted only 5 days.

On 2 occasions, the owner had dog 1's nose surgically explored at her own clinic. During the course of these procedures, the entire subcutaneous area of dog 1's right nasal area was meticulously examined, and no abnormalities or foreign bodies were found. During the second surgery, 3.8 cm of the maxillary division of the dog's trigeminal nerve was resected at the level of the maxillary foramen at the suggestion of a veterinary neurologist. Surgery was advised because carbamazepine did not alleviate the putative problem of trigeminal neuralgia.

In 1994, a veterinary neurologist at the Tufts University School of Veterinary Medicine reviewed a videotape of dog 1 performing the facial mutilation behavior and agreed with the other neurologists that the behavior was probably not related to seizure activity. In 1995, the dog was determined to have bilateral cataracts, and the left lens was removed by a board-certified ophthalmologist. Results of a CBC and serum biochemical analyses performed prior to this surgery were all within reference limits.

Dog 1 was also examined by a neurologist in 1995. Treatment with phenobarbital (15 mg, PO, q 12 h) was attempted but did not alter the dog's behavior. Treatment with phenobarbital was discontinued after 10 days, because the dog became extremely disorientated. The neurologist subsequently advised treatment with the anticonvulsant gabapentin (100 mg, PO, q 12 h, for 3 weeks). Again, there was no change in the frequency, intensity, or duration of the facial attacks.

In 1997, blood work was repeated, and radiography of the dog's skull and tympanic bullae was performed. No abnormalities were detected.

In 1998, dog 1 underwent extensive dental work, including extractions. Preoperative blood tests revealed increases in hepatic enzyme activities.

Diagnosis

During 1994, following written consultation with the Behavioral Service at the Tufts University School of Veterinary Medicine and review of a videotape of the dog's behavior, it was suggested that, providing all relevant medical conditions were ruled out, dog 1 may have a form of canine compulsive disorder.[1] In retrospect, it appears that this diagnosis was only partially correct. Although the environmental licking did seem to have a compulsive cause, the dog's facial attacking episodes later turned out to be more accurately described as attention-getting behavior.[2]

Treatment

As a result of the consultation with the Behavioral Service, dog 1's management was changed. Recommendations were to feed the dog a maintenance diet (as opposed to a performance-type diet), to increase the amount of exercise the dog received, and to continue to ignore bouts of the dog's scratching behavior. Additionally, dog 1 was treated with naltrexone (25 mg, PO, q 12 h) because of its known efficacy against self-directed behaviors.[3] However, there was no observable reduction in the frequency, intensity, or duration of dog 1's facial attacks after 3 to 4 weeks of this treatment.

Subsequently, dog 1 was treated with fluoxetine (7.5 mg, PO, q 24 h). After 3 months of this treatment, no major differences in the facial attacks were evident, but the dog's environmental licking behavior had lessened by about 30%, as assessed by daily observation of his routine behavior patterns. Propranolol (5 mg, PO, q 12 h), a β-adrenergic receptor blocker with $5HT_{1A}$ receptor blocking properties, was added to the treatment regimen to augment the effects of fluoxetine.[4,5] Again, no improvement was evident after 1 month of this treatment.

In 1995, treatment with clomipramine (12.5 mg, PO, q 24 h) was initiated, and the owner noticed a 50% reduction in the frequency of facial attacks. However, although the facial attacks were less frequent, the attacks initially seemed more intense, and the owner briefly considered euthanasia. Over time, the intensity of the facial attacks lessened somewhat, and the owner continued to treat the dog with clomipramine for approximately 3 years. Even though clomipramine was the only drug that consistently lessened the compulsive licking and reduced the intensity and frequency of the facial attacks, treatment with clomipramine was discontinued in April of 1998, when the dog was found to have hepatocellular carcinoma.

Follow-up

In November 1997, dog 2 died suddenly, and dog 1 was alone with the owner during the day, even though it still had some interactions with the other 2 dogs in the household during the evenings. After approximately 3 weeks, dog 1's owner realized that the dog had completely stopped the facial attacking behavior, suggesting that the facial attacks may have been a form of attention-seeking behavior, because the dog no longer performed them now that he was the primary subject of the owner's attentions.

A few months later, dog 1's owner rescued a Yorkshire Terrier pup that became dog 1's new companion, and the dog's facial attacks resumed approximately 6 weeks after the new arrival. This time, a nonpharmacologic behavior modification strategy of using a bridging stimulus (sounding a duck call) was employed to curtail the behavior. The duck call was used to focus dog 1's attention on a point in time, following which the owner would turn away or depart. The owner thought that this strategy did help, even though she was not able to use it consistently, and as previously, it was difficult to prevent strangers from responding to dog 1's facial attacks.

Shortly after this time, dog 1 was found to have high hepatic enzyme activities during routine serum biochemical testing performed prior to the dentistry.

Ultrasonography subsequently revealed a liver mass, and an exploratory laparotomy was later performed. The liver mass was surgically removed in 1998, when dog 1 was 14 years old. The mass was confirmed to be a hepatocellular carcinoma of low-grade malignancy. Dog 1 recovered from the surgery without complications and lived a fairly normal life for an additional 10 months, although intensity and frequency of the facial attacks gradually returned to their former levels.

In 1999, dog 1 was treated with butorphanol (3.75 mg, PO, q 12 h then q 8 h), a mixed opioid agonist-antagonist, to determine whether it would alleviate the facial attacks by virtue of its long-acting opioid receptor blocking activity.[3] Dog 1's owner indicated that frequency and intensity of the attacks decreased by 50% following treatment with butorphanol, but by this time, the hepatocellular carcinoma had recurred. The dog underwent a second surgery for excision of the cancerous mass, and 2 liver lobes were removed. Dog 1 survived another 6 months after the second surgery. During this time, the dog did not receive any treatment for the facial attacks, which continued in concurrence with compulsive licking of objects. The dog was eventually euthanatized because of end-stage liver cancer.

Discussion

The waxing and waning pattern of expression of dog 1's facial attacks seemingly depended on whether there were other dogs in the home, strongly indicating a psychological cause. Behaviorists would classify this behavior as attention-getting behavior,[2] occurring in what seemed to be a needy, anxious, and competitive dog. Certainly dogs can display a variety of complicated behaviors as a means of getting attention, as evidenced by dogs that feign lameness, develop coughing attacks or sneezing fits, or snap at imaginary flies to attract attention.[1] One of the authors has examined a dog that engaged in seizure-like behavior, assuming lateral recumbency and displaying clonic jerking movements with both hind limbs, to receive attention. Partial seizures were diagnosed by the referring veterinarian and a board-certified neurologist, but the behavior ceased completely after 3 weeks of use of a bridging stimulus and attention withdrawal. A wolf in captivity was described as displaying seizure-like behavior to solicit attention,[6] and the authors of that report ascribed the behavior to conditioning that occurred as a result of the attention the wolf received during treatment for seizures associated with organophosphate toxicosis.

What causes a particular attention-seeking behavior is not always clear. In dog 1's case, the facial attacking behavior may initially have arisen as a displacement behavior resulting from frustration, anxiety, and conflict. Alternatively, there may have been some medical reason, such as trauma or infection. By the time dog 1's owner realized there was problem, a tangible inciting cause was not evident, and the true initiating cause for the behavior will always remain a matter for speculation. Although complete withdrawal of attention turned out to be impossible in dog 1's case, when the dog became the subject of the owner's undivided attention, the facial attacks stopped completely. Even dog 1's licking behavior seemed to have an attention-seeking component, as this behavior worsened in situations during which the dog was denied full attention.

An earlier explanation for dog 1's facial attacks that was considered was that they were a form of neurotic excoriation.[7,8] This condition is well documented in humans and takes the form of constant scratching directed toward certain areas of the body, sometimes the face, often resulting in large excoriated areas. Attention seeking can hardly be an explanation for this behavior in human beings, who are usually extremely distressed at their inability to control themselves. One argument against neurotic excoriation being the cause of the dog's behavior was that there was no improvement during treatment with naltrexone, which has been found to be effective in humans with neurotic excoriation.[8] Dog 1's temporary partial response to butorphanol did reopen the possibility of neurotic excoriation, but the dog's complete response to receiving the full attention of its owner provides evidence in a different direction. The response to butorphanol may have had more to do with its sedative properties than to a specific anticompulsive effect.

Other possible reasons for dog 1's self-mutilation behavior include a form of canine compulsive disorder, a seizure-related disturbance, allergy, trigeminal neuralgia, dental disease, and hepatic dysfunction. Canine compulsive disorder was not definitively ruled out by the poor response of dog 1 to treatment with fluoxetine and clomipramine, because the dosages used, although considered optimal at the time, are now considered somewhat conservative. Also, fluoxetine was administered for too short a time for its effects to be properly evaluated. That the facial attacks ceased for a time following the death of dog 2 and resumed soon after another dog was obtained provides evidence against the theory that dog 1 had a compulsive disorder. Partial seizure activity was unlikely to be at the root of dog 1's behavior problem in light of the neurologists' clinical opinions and lack of positive response to 2 anticonvulsants (phenobarbital and gabapentin). It can be argued that treatment with phenobarbital was too brief to truly evaluate its effects, but some slight improvement may still have been expected. Allergy was another possible factor underlying the facial attacks. However, 5 veterinarians who examined dog 1 did not regard the behavior to be a result of a dermatologic disorder, as there were no cutaneous manifestations of allergy and no other findings typically associated with allergy. Also, unilateral expression of allergy would have been unusual. Dog 1's apparent partial response to treatment with prednisolone acetate may have been accountable as a central effect[9] or may have been artifactual or coincidental. Trigeminal neuralgia was an interesting and credible possible diagnosis, but targeted drug treatment and neurectomy failed to alleviate the condition. Likewise, dental treatment failed to alleviate the condition, pointing against a dental cause. Although hepatic dysfunction must have affected dog 1 later in life, results of laboratory tests, including tests of liver function, were normal when the behavior first started, and hepatic enzyme activities were measured several time between 1992 and 1997 and were normal.

The ancillary diagnosis in this dog was compulsive licking of objects in the environment. Compulsive licking of objects is 1 way in which canine compulsive disorders can express themselves.[1] The fact that dog 1's licking behavior responded so well to treatment with clomipramine is supportive of this diagnosis, as clomipramine has potent anticompulsive properties.[5] Medically, compulsive behaviors are now classified as anxiety-related disorders.[10] Dog 1's sensitive personality fits well with this secondary diagnosis and with our conclusion that the facial attacks were propagated largely as an attention-seeking behavior.

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