CASE IN POINT

REPETITIVE CHEWING & PAWING AT THE MOUTH IN A CAT

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Brady, a 13-year-old neutered male Ragdoll cat, was presented for once- to twice-weekly episodes of repetitive chewing and pawing at the left side of the mouth of varying duration (typically 5-10 minutes) that began approximately a month prior to presentation.

The episodes always had a sudden onset and most commonly occurred immediately or within a few minutes after eating but also occurred spontaneously without an obvious trigger. The owner was sometimes able to briefly distract the cat out of the episodes. Brady was normal between episodes and had no postepisode abnormalities. He was an indoor-only cat with no known past or recent trauma or medical history. There was one other cat in the household.

Initial Presentation

On initial presentation to his primary care veterinarian, Brady had grade IV periodontal disease with some missing teeth that were thought to be resorptive lesions, but the specific details were not described in the record. The remainder of the physical examination was unremarkable.

CBC, serum chemistry profile, and total thyroid hormone were within normal limits except for mild nonregenerative anemia (hematocrit, 28%; range, 30%-45%), renal azotemia (BUN, 37 mg/dL; range, 16-35 mg/dL; creatinine, 2.9 mg/dL; range, 0.8-2.4 mg/dL), and isosthenuric urine (specific gravity, 1.009; range, 1.008-1.060).

Dental radiography revealed resorbed tooth roots, but the specific findings were no longer available in the referring veterinarian's records. The primary care veterinarian extracted the teeth with resorptive lesions, but the owner reported no improvement in the episodes following dental treatment, so the cat was referred for neurologic evaluation.

Neurologic Evaluation

General physical and neurologic examinations were within normal limits. A video of one of Brady's episodes was provided by the primary care veterinarian and showed repetitive chewing movements, pawing at the face, and signs of discomfort (see ***Video***). During the episode, Brady appeared distressed but without altered mentation. Differential diagnoses included feline orofacial pain syndrome (FOPS), complex psychomotor seizures, dental disease, temporomandibular joint disease, otitis media, and abscess/cellulitis. Seizures were considered less likely due to the patient's normal consciousness during the episodes, the ability to distract the cat from the episodes, and the lack of postictal signs. There were no signs of pain on opening of the mouth, and no obvious abnormalities (eg, tooth root abscess, uremic lesions secondary to chronic kidney disease) were noted during sedated oral examination, so an oral or temporomandibular disorder was considered less likely.

MRI of the head was recommended to rule out structural causes, but the owner elected to pursue empiric treatment due to financial constraints.

DIAGNOSIS:
FELINE OROFACIAL PAIN SYNDROME (FOPS)

Long-Term Management

Brady's primary differential diagnosis was FOPS. Because FOPS is a diagnosis of exclusion and the owner had financial constraints, the owner elected to pursue empiric treatment. Gabapentin

VIDEO

Scan the QR code to watch the video of Brady's episode.

Using QR codes from your mobile device is easy and quick!

Simply focus your phone's camera on the QR code as if taking a picture (but don't click!). A notification banner will pop up at the top of your screen; tap the banner to view the linked content.

BMS = burning mouth syndrome
FOPS = feline orofacial pain syndrome

(10 mg/kg PO q8-12h) was prescribed; NSAID therapy was not initiated due to the cat's renal azotemia. Administration of gabapentin led to a significant decrease in the frequency and severity of episodes, reducing them from once or twice weekly to once every 4 to 6 weeks. Brady was maintained on gabapentin and did well long-term but was euthanized 2 years later due to an unrelated disorder.

Discussion

Only one large-scale retrospective study has been published on FOPS, an enigmatic condition characterized by episodes of exaggerated licking, chewing movements, and pawing at the mouth.[1,2] Ulceration of the tongue, lips, and buccal mucosa can occur in severely affected cats.[1,2] The cause has not been identified, but FOPS is generally thought to be the result of neuropathic pain isolated to the face and mouth.[1,2] Episodes can vary in duration and frequency, ranging from minutes to hours. Distracting the cat temporarily out of the episodes is sometimes possible.[1,2] Inappetence and weight loss may be noted in some patients.

FOPS shares some clinical features of 2 neuropathic facial pain syndromes in humans, trigeminal neuralgia and glossodynia. Trigeminal neuralgia is characterized by brief episodes of severe pain in areas innervated by the trigeminal nerve, particularly the mandible and maxilla.[3,4] Because episodes are often preceded by mild sensory stimulation (eg, light touch, shaving, wind currents, eating/drinking) that would not normally provoke a painful response, trigeminal neuralgia can be characterized as a form of allodynia.[3] Demyelination of sensory fibers in the trigeminal nerve has been observed in the nerve root and, less commonly, the brainstem of affected patients. Most trigeminal neuralgia cases result from compression of the CNS portion of the trigeminal nerve root by an overlying artery or vein,[3,4] although secondary (ie, noncompressive) trigeminal neuralgia is possible and has been reported with several neurologic disorders, including multiple sclerosis and other myelinopathies, surgical trauma, neoplasia, and trauma.[4] Microvascular decompression surgery is recommended for compressive trigeminal neuralgia patients that are refractory to medical management and often provides rapid relief of clinical signs.[3,4] Neuroablative techniques (eg, stereotactic radiosurgery, radiofrequency thermocoagulation) are recommended in refractory patients that are unable to undergo microvascular decompression and in patients with noncompressive trigeminal neuralgia; idiopathic disease should be considered when there is no evidence of neurovascular contact.[4] As with trigeminal neuralgia, FOPS episodes are frequently induced by nonnoxious stimuli (eg, eating, drinking). Compression of the trigeminal nerve has not been reported as a cause of FOPS.

Glossodynia (ie, burning mouth syndrome [BMS]) is a condition in humans in which patients experience distortion in taste perception (ie, dysgeusia), dry mouth (ie, xerostomia), psychosocial distress, and a burning, tingling, or prickling sensation of the oral mucosa, particularly on the tip of the tongue (ie, glossodynia).[5-7] BMS is characterized by a lack of physical abnormalities detected on oral examination.[5-7] The etiology is not entirely clear, but it appears to be multifactorial. Sex hormone dysregulation is considered an important

TREATMENT AT A GLANCE

- Anti-inflammatory (eg, NSAIDs, prednisone) and pain medications (eg, gabapentin, pregabalin) are the most commonly prescribed treatments in patients with FOPS.
- Dental treatment is recommended in FOPS patients if periodontal disease is present.
- Anticonvulsants with antiallodynic properties (eg, phenobarbital, topiramate) or antidepressants (eg, amitriptyline, fluoxetine) may be needed for refractory patients.
- Prevention of self-mutilation and reduction of psychosocial stress are important nonpharmacologic therapies.

causative factor, as the condition is more common in peri- and postmenopausal women.[5] Historically, BMS has been considered to be psychosomatic, as many BMS patients are predisposed to anxiety and/or depression.[5,8-10] However, although psychosocial events can trigger or exacerbate symptoms, BMS can also be the cause of—rather than induced by—anxiety and depression.[5,8-10] There is no cure for BMS, but medications commonly used to manage BMS include neuroprotective steroids, hormone replacement therapy, anxiolytics (eg, clonazepam), and selective serotonin reuptake inhibitors (eg, fluoxetine) or serotonin and noradrenaline reuptake inhibitors.[5,8-10] As with glossodynia, FOPS episodes may be triggered or exacerbated by psychosocial stress.

A strong breed predilection for FOPS has been identified in Burmese cats and related crossbreeds; in a prospective study, 88% of FOPS cases were Burmese cats or related crossbreeds.[1,2] Other reported breeds include domestic shorthair, Siamese, British shorthair, and Somali cats.[1,2] Pedigree analysis has suggested that FOPS may be hereditary in the Burmese breed, but insufficient data exist to identify the mode of transmission.[1]

Mean age of onset is ≈7 years, but age is not normally distributed.[1,2] In the previously mentioned prospective study, there was a peak age of onset in immature cats, with 19 (17%) cats <6 months of age at the time of the first FOPS event.[1,2] In many of the immature cats, episodes were related to teething and other dental changes.[1,2] After removing the immature cats from the dataset, the age distribution curve was normally distributed. Eating, drinking, and grooming were commonly reported triggers.[1,2] Anxiety was reported as a possible trigger in 24 (21%) cats; signs of anxiety included social incompatibility in multicat households (eg, introduction of a new cat), moving, death of a caregiver, veterinary hospitalization, and other psychosocial stressors.[1,2]

Neurologic examination is usually normal between episodes but may show evidence of abnormal facial sensation or jaw tone.[1,2]

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Physical examination may reveal evidence of self-mutilation of the face, mouth, and/or tongue. In the prospective study, ≈63% of the cats had oral disorders (eg, eruption of permanent teeth, dental disease, oral ulceration) at the time of diagnosis.[1] Neurologic examination is usually normal between episodes but may show evidence of abnormal facial sensation or jaw tone.[1,2]

A presumptive diagnosis is generally made based on clinical signs, particularly in the presence of identifiable triggers or risk factors, and on the exclusion of other potential causes (eg, complex psychomotor seizures, oral foreign body, trigeminal nerve sheath tumor or lymphoma, trauma).[11,12] CBC, serum chemistry profile, and thyroid hormone screening should be performed to rule out concurrent metabolic and/or systemic disease, and sedated oral examination and dental radiography should be performed to evaluate for dental disease. In the prospective study, MRI results were normal in the 2 cats in which MRI was performed, but MRI should be still considered in patients in which FOPS is suspected to rule out structural causes, particularly if any abnormalities are detected on neurologic examination (see ***Take-Home Messages***).[1]

Oral disease should be identified and treated appropriately. Approximately 50% of the cats in the prospective study that underwent dental treatment had sustained improvement in clinical signs.[1] However, a direct association between the presence of dental

disease and FOPS is difficult to prove, as there is a high incidence of periodontal disease in cats without evidence of FOPS (≤72% in purebred cats).[1,13]

Treatment should be centered on relieving pain, preventing self-mutilation, and reducing environmental factors (see ***Treatment at a Glance***, page 21). First-line treatment typically includes NSAIDs (eg, meloxicam) or pain medications (eg, gabapentin, pregabalin, opioid).[1,2] Gabapentin has been shown to be effective in the treatment of buccofacial allodynia and trigeminal neuralgia in humans.[14,15] Prevention of further self-mutilation (eg, Elizabethan collar, nail caps, paw bandaging) and reduction of psychosocial distress (eg, providing secluded areas, additional litter boxes, and pheromone diffusers) are also important treatment components.[1,2] In refractory patients, a trial course of an anticonvulsant with antiallodynic properties (eg, phenobarbital, levetiracetam, topiramate) or an antidepressant (eg, amitriptyline, fluoxetine) should be considered.[1]

The prognosis for FOPS is fair to good. Spontaneous remission may occur in some cats, but most require treatment. In the prospective study, ≈50% of cats with FOPS responded to short-term medications during episodes[1,2]; some were weaned off medications entirely or between recurrent bouts.[1,2] Approximately one-third of cats required long-term treatment, and ≈10% were euthanized due to the severity of clinical signs.[1,2]

TAKE-HOME MESSAGES

- FOPS is characterized by episodes of exaggerated licking, chewing movements, and pawing at the mouth.
- It is important to differentiate FOPS episodes from other paroxysmal episodes, particularly complex psychomotor seizures with orofacial involvement.
- Advanced imaging (MRI preferred over CT) should be considered in all patients with clinical signs of FOPS to rule out structural causes of the episodes, particularly if there is no evidence of dental disease or if neurologic abnormalities are noted on examination.
- Many cats respond to treatment with anti-inflammatory and pain medications, but anticonvulsants with antiallodynic properties or antidepressants may be needed for refractory patients.

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