Not Just a Pretty Smile: Neurologic Disorders of the Head and Face

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Neurologic diseases affecting the structures of the head are a common clinical presentation in veterinary practice. A review of the most common disorders will be discussed during the presentation in a case-based review format.

Trigeminal neuritis is an idiopathic, bilateral, nonsuppurative inflammation of the motor branches of cranial nerve V. It has been reported in dogs and cats and the pathogenesis remains unknown. The clinical picture is an acute onset of jaw paralysis with an inability to close the jaw. Most patients remain able to swallow, although mild dysphagia may be seen. Other findings including Horner's syndrome, facial sensory deficits and muscle atrophy are less commonly seen. A tentative diagnosis is made based on the clinical features. EMG and MRI imaging may be useful to rule-out other differentials including nerve sheath tumours in unilateral nerve involvement. Lymphosarcoma and Neospora have also been reported with similar presenting signs. This is a self-limiting disease and most patients recover within 2-3 weeks. There is no evidence that corticosteroid therapy is effective in promoting recovery. Some patients may require nursing care including hand-feeding during the recovery period.

Idiopathic facial paralysis is an acute mononeuropathy of one or both facial nerves. This condition has been reported in dogs and cats and also has an unknown pathogenesis. Bell's palsy is a similar condition that occurs in people. There is some evidence Bell's palsy is due to an immune-mediated response triggered by the herpes simplex virus. Affected animals are usually middle-aged to older, and numerous breeds have been reported. Cocker Spaniels appear to be overrepresented. Common clinical findings are expected signs of facial nerve dysfunction (drooping of the ears and lips, deviation of the nasal philtrum, decreased to absent palpebral reflex). Corneal ulceration may occur due to inadequate blinking. Vestibular dysfunction is uncommonly seen in some patients. Diagnosis is based on characteristic historical and presenting signs. Other conditions such as otitis and hypothyroidism should be ruled-out. The prognosis is guarded for complete return of function of the facial nerve. Full recovery may occur in weeks to months following the onset of signs. Treatment is symptomatic (e.g, artificial tears to prevent corneal ulceration). Corticosteroid therapy is controversial and likely does not influence the recovery.

Masticatory muscle myositis is an autoimmune disorder in which antibodies are directed against the muscles of mastication (e.g, temporalis, masseter, pterygoid mm.), with a unique myosin isoform (type IIM). Dogs of numerous breeds and both sexes have been reported, but the

German Shepherd dog appears to be predisposed. A juvenile onset in dogs as well as several cats have been rarely reported. Clinical signs are often acute and typically include painful swelling of the masticatory muscles as well as trismus. Palpation of the masticatory muscles and attempts to open the jaw often elicit a pain response. Diagnosis is based on the demonstration of antibody localized to type IIM myofibers. This can be performed using frozen sections of the patient's temporalis muscle or serum. The test has high sensitivity and specificity, but may yield false-negative results in a small subset of dogs with MMM. CK levels may be elevated and EMG exam is often abnormal. CT scan and MRI imaging may also aid in the diagnosis, showing contrast enhancement, muscle size changes, or attenuation in the masticatory muscles. Treatment is immunosuppressive doses of prednisone (1-2mg/kg PO BID) for 3-4 weeks. A slow and tapered dose to the lowest every-other-day dosage that will control clinical signs is then recommended. Most dogs show a favourable response, although some may require other maintenance immunosuppression (e.g., azathoprine). The prognosis is generally favorable, although reduced function and muscle atrophy may persist.

Cavernous sinus syndrome (CSS) is a rare clinical disorder in dogs (and cats) characterized by dysfunction of many of the cranial nerves (CNs) that course in intimate association with the cavernous sinuses, including the oculomotor nerve (CN III), trochlear nerve (CN IV), abducens nerve (CN VI), and the maxillary and ophthalmic branches of the trigeminal nerve (CN V). Because CN III, CN IV, and CN VI provide innervation to the extraocular musculature and the parasympathetic efferent fibers of CN III are responsible for pupillary constriction, the most common clinical manifestations of CSS are external and internal ophthalmoparesis, ptosis, and mydriasis. Because postganglionic sympathetic nerve fibers also lie in close proximity to the cavernous sinus, CSS can also be associated with signs of oculosympathetic denervation. Diagnostic imaging studies of the brain and orbits are usually needed to differentiate CSS from retrobulbar diseases involving the orbital fissure, which can cause similar clinical signs. Pathogenesis of CSS commonly results from neoplastic infiltration of the cavernous sinuses. However, trauma, vascular anomalies, and inflammatory diseases have also been implicated as potential causes of CSS in both species. Results of a recent study suggest that bilateral CSS is rare in dogs but that a mass lesion involving the cavernous sinuses should be suspected in dogs with clinical evidence of concurrent dysfunction of CN III, CN IV, CN VI, and the first 2 branches of CN V, with or without evidence of oculosympathetic denervation, once retrobulbar disease has been adequately ruled out. In 5 of the 6 dogs in this study, bilateral CSS was a result of neoplasia (the underlying cause was not determined in the remaining dog), suggesting that neoplasia should be the primary diagnostic consideration in dogs with bilateral CSS, particularly in dogs with concurrent physical abnormalities and neurologic deficits referable to an extra-sinusoidal region. Ultrasonography, computed tomography, and magnetic resonance imaging were useful in characterizing the lesions causing CSS in these dogs. All dogs had advanced clinical signs at the time of initial examination and were eventually euthanized because of progressive disease.

References:

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