Neurology Neurologie

Unilateral vestibular disease

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Clinical case

A 19-year-old castrated, male domestic long-haired cat was referred to the Western Veterinary Specialist Centre with a history of an acute onset of leaning and falling, a horizontal nystagmus with the fast phase towards the right, and seizure-like episodes that had been occurring for approximately 1 wk duration. A complete blood (cell) count, serum biochemistry, urinalysis, and resting tetraiodothyronine (T4) were performed before referral and were deemed unremarkable.

Upon examination, the cat was lethargic and inappetent. Distant examination revealed that the cat was thin and had an obvious head tilt toward the left. When permitted to walk freely in the examination room, the cat would lean toward the left. Physical examination, including thoracic and abdominal auscultation, palpation of the head, neck, limbs and abdomen, and an aural examination, was unremarkable. Neurological examination, including assessment of mentation, examination of the cranial nerves, spinal reflexes, and postural responses, revealed that the cat had an appropriate/clinically normal mental status given its history, age and species, though it had a left-sided head tilt, absent left-sided palpebral reflex and menace response. The previously reported pathological nystagmus was not present. Indirect ophthalmoscopic examination of the ocular fundi was unremarkable.

What is your neuroanatomic diagnosis?

a)   left-sided peripheral vestibular system
b)   right-sided peripheral vestibular system
c)   left-sided central vestibular system
d)   right-sided central vestibular system
e)   cerebellum

Case discussion

Our neuroanatomic diagnosis was a) a disease process affecting the left peripheral vestibular system, specifically the left inner (for the head tilt and vestibular signs) and middle ear (for the facial nerve deficit). In order to accurately neuroanatomically localize where a disease process is occurring it is important to be knowledgeable about the neuroanatomy and the function of that anatomy.

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The ear is divided into the outer ear, middle ear, and inner ear (1,2). The outer ear is made up of the pinna, the vertical and horizontal ear canal, and is bordered proximally by the tympanic membrane (1,2). The middle ear is an air-filled space containing 3 bony ossicles (malleus, incus, and the stapes), is contained within the tympanic bulla, and is directly connected with the nasopharynx via the auditory (Eustachian) tube (1,2). The post-ganglionic sympathetic axons of neuronal cell bodies arise from the cranial cervical ganglion, cross at the roof of the middle ear and adjacent to the inner ear, before traveling rostrally to innervate the iris of the eye (1). Cranial nerve VII (facial nerve) also traverses near the middle ear, and for a short distance, with cranial nerve VIII (vestibulocochlear nerve) (1). Consequently, surgery, or disease processes that affect the middle and inner ear may cause vestibular signs (related to inner ear involvement), Horner's syndrome (enophthalmos, miosis, ptosis, and protrusion of the third eyelid) and facial nerve paralysis (related to middle ear involvement) (3,4).

The inner ear is made up of the bony labyrinth that contains the membranous labyrinth within the petrous portion of the temporal bone of the skull (1,2). The inner ear has dual function in that the membranous labyrinth is made up of both the vestibular and hearing portions. The hearing portion of the membranous labyrinth is known as the cochlea, while the vestibular portion is comprised of the semicircular canals (responsible for detecting rotational movement), and the saccule and utricle (responsible for detecting linear movement) (1). Afferent (sensory) vestibular axons join the afferent axons coming from the cochlea and enter the cranial vault as cranial nerve VIII (vestibulocochlear nerve) via the internal acoustic meatus (1). Cranial nerve VIII enters the cranial vault with the facial nerve, and penetrates the brain at the level of the rostral medulla oblongata (1). Vestibular afferent axons synapse with neurons located in the vestibular nuclei and with neurons within the cerebellum (5). The vestibular nuclei will send projections to many regions of the brain (5). In particular, vestibular nuclei make connections with cranial nerves responsible for the functioning of ocular movements (Cranial nerves III, IV, and VI) via a collection of axons (tract) known as the medial longitudinal fasciculus (1). Thus, damage to the vestibular system can account for any observed strabismus or pathological nystagmus. It should also be noted that the vestibular nuclei lie amongst a network of neurons responsible, in part, for arousal and motivation. This network is made up of the reticular formation and its connections [clinically functionally known as the ascending reticular activating system (ARAS)]. Given that the ARAS extends along the length of the brainstem, and that disease processes affecting

Table 1. Clinical signs associated with unilateral vestibular disease

| Vestibular syndrome	Mentation	Cerebellar signs	Ataxia	Head tilt and body falling/rolling	Spontaneous nystagmus	Involvement of other cranial nerves	Horner's syndrome	Proprioceptive deficits and paresis
Peripheral	Appropriate for a clinically normal animal (consider history, age, breed, and species)	-	+	+, toward the lesion	+, (horizontal or rotary and fast phase away from side of lesion) — acute stage	+/-, (same side as the lesion; CN VII)	+/-	-
Central (nonparadoxical)	Usually inappropriate (such as, lethargic, somnolent)	-	+	+, toward the lesion	+, (horizontal, rotary or vertical, fast phase away from lesion)	+/-, (same side as the lesion; CN V, VI, VII)	-	+/-, ipsilateral to the lesion
Central (paradoxical)	Usually inappropriate (such as, lethargic, somnolent)	+/-	+	+, away from the lesion	+/-, (horizontal, rotary or vertical, fast phase away from lesion)	+/-, (same side as the lesion; V, VI, VII)	-	+/-, ipsilateral to the lesion
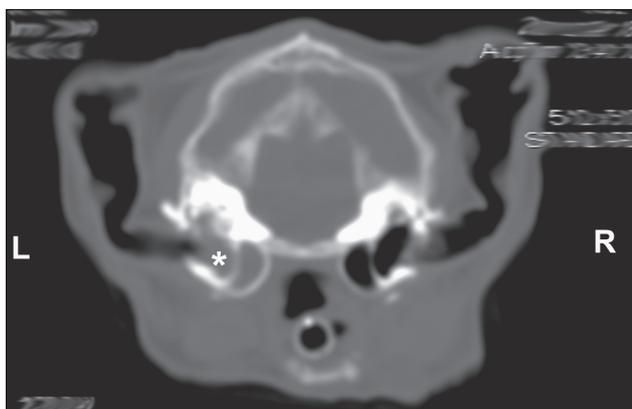
Figure 1. Transverse computed tomographic view of the cat's head (bone window). Note the radiodense appearance of the left auditory bulla (asterix) compared to the clinically normal appearing right bulla.

the central vestibular system are situated in this region, animals with central vestibular disease will often have clinical signs suggesting involvement of the ARAS (quiet demeanor, lethargy, change in mentation, etc).

Table 1 summarizes the clinical signs used to neuroanatomically localize unilateral disease processes to the peripheral or central portion of the vestibular system. It should be noted, however, that experimentally induced unilateral vestibular disease appears very similar to the clinical situation and that, like the clinical situation, vestibular compensation of static responses (such as, spontaneous nystagmus and head tilt) occurs as was seen in the present case (seemingly spontaneous resolution of nystagmus) (6). Vestibular compensation results from a variety of proposed mechanisms that involve long-lasting or permanent neuroanatomical and biochemical changes of the nervous system resulting from some prior experience, which is also known as neuroplasticity. Nevertheless, once an accurate neuroanatomic diagnosis has been made, one can begin to determine an appropriate list of differential diagnoses, thus facilitating the creation of a diagnostic plan.

There are various conditions that cause vestibular disease in feline species (7,8). Common conditions affecting the peripheral vestibular system include, idiopathic vestibular disease (9), otitis media-interna (8), trauma (8), toxins (such as, aminoglycosides; propylene glycol, chlorhexidine, and cetrimide-containing topical ear cleansers) (10,11), nasopharyngeal polyps (12,13), neoplasia (12,14), and a suspected inherited congenital peripheral vestibular disorder (8). Given the cat's signalment and history and our clinical findings, our differential diagnoses were neoplasia, idiopathic geriatric vestibular disease, or an inflammatory or infectious disease process such as nasopharyngeal polyp or otitis media-interna. A recommendation to perform a computed tomography (CT) examination of the cat's head was made. While the cat was under anesthesia for CT examination, complete otoscopic and oral examinations were performed and these were unremarkable. The CT examination (GE HiSpeed CT/i) revealed that the left tympanic bulla was isodense compared with the surrounding soft tissue (Figure 1) and was considered to be consistent with fluid and/or soft tissue occupying the normally air-filled tympanic bulla. Before recommending aural surgery for diagnostic and therapeutic reasons, a 3-view thoracic radiograph was performed to ensure there was no evidence of metastasis, should the mass in the left middle ear have been neoplastic. Thoracic radiographs were unremarkable. The cat then underwent left lateral bulla osteotomy and abnormal tissue and fluid were removed from the left middle ear and were submitted for histopathologic examination and bacterial culture and sensitivity. The cat recovered uneventfully and was prescribed systemic amoxicillin-clavulanic acid (Clavamox Drops; Pfizer Canada, Quebec), 13.5 mg/kg, PO, BID, until results of bacterial culture and antimicrobial sensitivity became available. A nonsteroidal anti-inflammatory drug [Meloxicam; Metacam, Boehringer Ingelheim (Canada), Ontario] was administered for 2 d and a 1/2 of a 25 μg fentanyl patch (Ratio-fentanyl 25 μg; Ratiopharm, Toronto, Ontario) was administered transdermally over the thorax for 5 d so as to control post-operative pain. In addition, meloxicam, 0.1 mg/kg, PO, q24h for 2 d was administered for post-operative pain relief. Histopathology of the tissue provided a diagnosis of chronic-active, mixed cellular otitis

media with evidence of polypoid hyperplasia; no bacteria were detected by histopathology. A diagnosis of feline inflammatory polyp or nasopharyngeal polyp was made. Bacterial culture resulted in no growth of bacteria. The cat continued to recover well 3 wk later as was evident by the cat becoming appetent again, although a mild head tilt persisted.

Nasopharyngeal polyps in cats are the most common benign pharyngeal and external/middle ear masses observed in this species (for review see reference 12). Nasopharyngeal polyps are typically found in cats that are less than 3 y of age, though the condition can be observed at any age. Polyps may be unilateral or bilateral, and affected animals typically have upper respiratory signs when the mass is located in the nasopharyngeal region or signs consistent with a disease affecting the middle and external ear. The etiopathogenesis of this condition is unknown, although several hypotheses have been proposed including suspected congenital origin in some cats, chronic upper respiratory infection or otitis externa/media, and viral infectious etiologies [such as, feline herpes virus-1 or calicivirus (15)]. As in the present case, treatment of nasopharyngeal polyps involves surgical removal of the polyp if it is in the middle ear or by simple traction avulsion if the polyp is solely within the mouth or the external ear canal. The removed mass should always be submitted for histopathological examination for a definitive identification. When middle ear involvement is present, tissue and/or fluid samples are submitted for bacterial culture and sensitivity as a bacterial otitis media may also be present. Prognosis for this condition after surgical removal is excellent when the polyp has been completely removed.

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