Unilateral Laryngeal Paralysis Secondary to Otitis Media/Interna in Two Cats

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ABSTRACT

Two adult cats were presented for coughing, gagging, dysphonia, exaggerated swallowing attempts, unilateral vestibular dysfunction, and/or Horner syndrome. In both cats, unilateral laryngeal paralysis was identified on the side ipsilateral to other neurological deficits. Cross-sectional imaging was consistent with otitis media/interna. In both cats, there also was extensive cellulitis surrounding the tympanic bulla and dissecting through tissue planes to involve the opening of the tympano-occipital fissure on the side ipsilateral to the laryngeal paralysis. Laryngeal paralysis was presumed secondary to involvement of the vagus nerve as it emerged from the tympano-occipital fissure. Antibiotic therapy resulted in resolution of clinical signs in both cats and restored laryngeal function as evidenced by visual examination of the larynx in one cat. (*J Am Anim Hosp Assoc* 2022; 58:42–47. DOI 10.5326/JAAHA-MS-7099)

Introduction

Laryngeal paralysis occurs infrequently in cats. Clinical signs of laryngeal paralysis in cats include dyspnea, tachypnea, dysphonia, gagging/retching, cough, and exercise intolerance.[1–5] Insufficient cases have been reported to define sex, neuter status, or breed predilections. Most affected cats have bilateral laryngeal paralysis.[1,4–6] In cats with unilateral laryngeal paralysis, the left side is more commonly affected.[4–6] In most cases, an underlying etiology is not determined and presumed idiopathic.[2,5,6] A congenital disorder is presumed in cats <2 yr old when an alternative explanation is not identified.[6,7] Acquired causes of laryngeal paralysis in cats include lymphoma of the vagus nerve, mediastinal neoplasia, infiltrative laryngeal neoplasia, postoperative thyroidectomy, cervical trauma, and generalized neuromuscular disease.[2,6,8–10]

The following case report describes unilateral laryngeal paralysis in two cats with otitis media/interna (OMI). In both cats, the onset of clinical signs of laryngeal paralysis shortly preceded the identification of neurological signs related to OMI including ipsilateral vestibular dysfunction and Horner syndrome. MRI of the head revealed extensive cellulitis of the tissues external to the tympanic bulla that extended caudally to involve the emergence of the vagus nerve from the tympano-occipital fissure. To the authors' knowledge, this is the first description of laryngeal paralysis secondary to OMI in the cat.

Case Report

A 6 yr old (3.6 kg) spayed female domestic shorthair (cat 1) was evaluated for coughing, gagging, and Horner syndrome in the right eye (OD). One month prior, the cat displayed episodes of coughing and gagging. Three weeks later, miosis OD and protrusion of the third eyelid developed. Physical examination performed by the referring veterinarian was normal with the exception of Horner syndrome OD. Complete blood count revealed lymphopenia (1.35×10^3; reference range $1.5–7.0 \times 10^3$ cells/L). Serum biochemistry revealed hyperglycemia (169 mg/dL; reference range 70–150 mg/dL), hyperproteinemia (9.4 g/dL; reference range 5.4–8.2 g/dL), and

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CN (cranial nerve); CT (computed tomography); OD (right eye); OMI (otitis media/interna); T1W (T1-weighted); T2W (T2-weighted); VBO (ventral bulla osteotomy)

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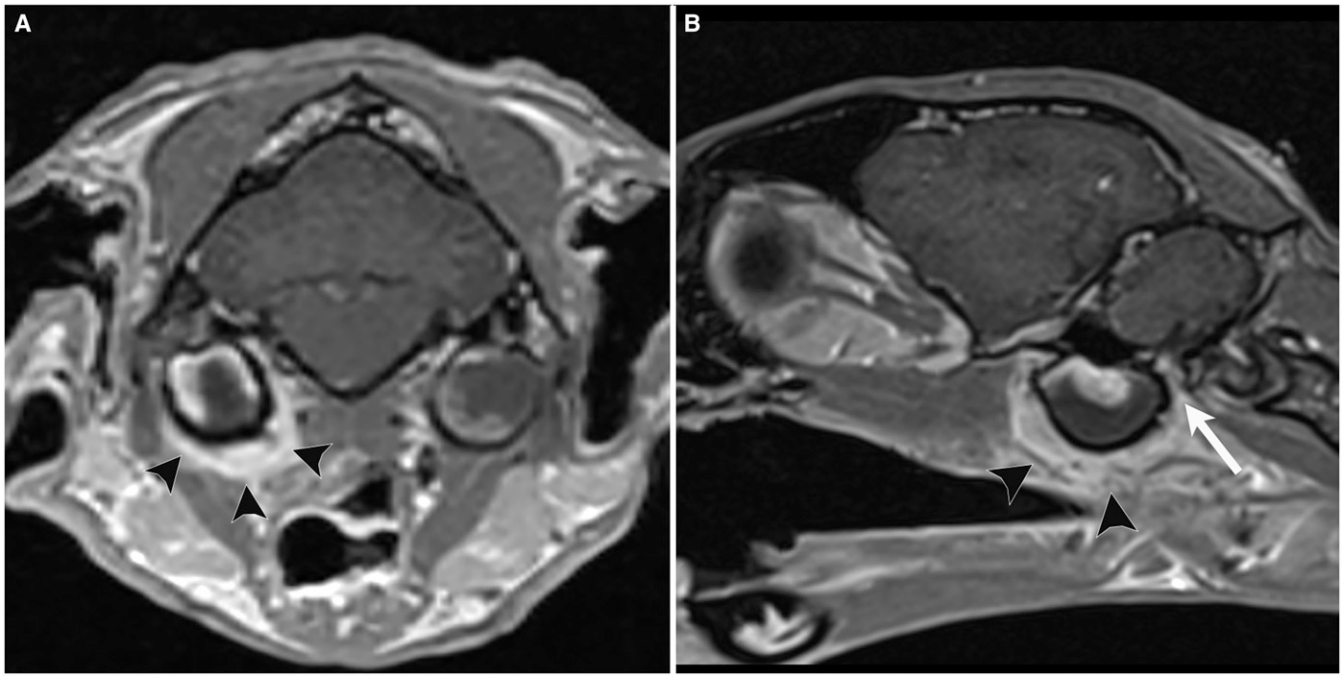

FIGURE 1

Magnetic resonance images of a 6 yr old cat with unilateral laryngeal paralysis and Horner syndrome. On the transverse reconstructed image (A) *at the level of the caudal aspect of the tympanic bulla and sagittal image* (B) *of the postcontrast T1-weighted, volumetric interpolated breath-hold examination sequence in cat 1* (A)*, strongly contrast-enhancing tissue* (black arrowheads) *surrounds the external surface of the tympanic bulla on the side ipsilateral to laryngeal paralysis. The enhancing tissue extends into the region of the tympano-occipital fissure* (white arrow).

hyperglobulinemia (6.1 g/dL; reference range 1.5–5.7 g/dL). The cat was prescribed robenacoxib[a] (1.6 mg/kg, orally *q* 24 hr). One week later, signs persisted and the cat was referred to the Neurology service at the Veterinary Teaching Hospital, University of Georgia, for further evaluation. The cat had no prior medical history of ear disease, was negative for feline leukemia virus and feline immunodeficiency virus, and was up to date on vaccinations.

On presentation, physical examination was normal with the exception of the ears. On an awake otoscopic examination, the right tympanum was opaque and thickened. A ceruminolith obscured visualization of the left tympanum. Neurologically, the cat had normal mentation, gait, postural reactions, and spinal reflexes. Cranial nerves (CNs) were normal with the exception of Horner syndrome OD. The cat was sedated with ketamine[b] (4.8 mg/kg) and midazolam[c] (0.25 mg/kg) IV for computed tomography (CT) of the head, neck, and thorax. Transverse images were acquired with a standard acquisition protocol through use of a 64-slice multidetector row CT unit[d]. Images were acquired in helical mode at a slice thickness of 2.0 mm using a tube voltage of 120 kVp, a tube current of 250 mAs, a tube rotation time of 0.5 s, and pitch of 0.8. Convolution algorithms for both soft tissue and bone were applied to the multislice data set. Images were acquired before and after administration of contrast medium[e] (600 mg/kg IV).

On CT, both tympanic cavities and the left horizontal ear canal were filled with non–contrast-enhancing fluid to soft tissue–attenuating material. The right tympanic bulla was enlarged and had punctate lysis and irregular periosteal proliferation. In the right nasopharynx, there was a 10.0 mm × 5.0 mm × 5.0 mm, soft tissue–attenuating mass that displayed peripheral contrast enhancement. The neck and thorax were normal. The CT findings were consistent with left-sided otitis externa and bilateral otitis media. The soft tissue mass in the nasopharynx likely was a nasopharyngeal polyp.

Twenty-four hours later, the cat was sedated for a laryngeal exam followed by MRI of the head. For laryngeal examination, the cat was sedated with butorphanol[f] (0.2 mg/kg IV) and propofol[g] (4 mg/kg IV given to the effect of sedation that enabled visualization of the larynx without causing apnea). On laryngeal examination, there was right-sided laryngeal paralysis. Following the laryngeal examination, additional propofol was given to induce general anesthesia for MRI of the head.

MRI was performed using a 3.0 T MRI unit[h] and a multichannel extremity coil. Multiplanar images of the head were acquired

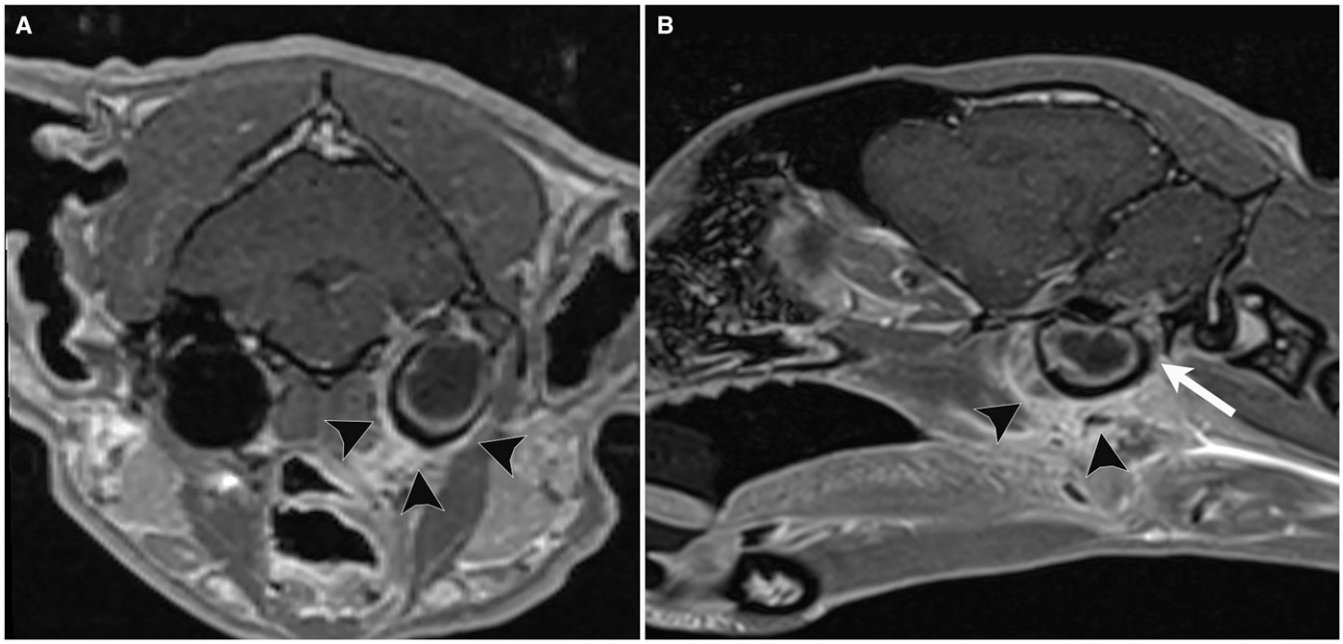

FIGURE 2

Magnetic resonance images of a 7 yr old cat with unilateral laryngeal paralysis, peripheral vestibular dysfunction, and partial Horner syndrome. On the same images and sequence as in Figure 1, the transverse image (A) *at the level of the caudal aspect of the tympanic bulla and sagittal* (B) *images show similar strongly contrast-enhancing tissue that surrounds the external surface of the tympanic bulla* (black arrowheads) *and extends into the region of the tympano-occipital fissure* (white arrow) *on the side ipsilateral to laryngeal paralysis in cat 2.*

using the following sequences: T2-weighted (T2W), T2W fluid-attenuated inversion recovery, T1-weighted (T1W), and susceptibility-weighted sequences. Following administration of gadopentetate dimeglumine[i] (0.2 mmol/kg IV), T1W and three-dimensional spoiled gradient echo (T1-volumetric interpolated breath-hold examination) sequences were acquired.

Bilaterally, the tympanic cavities were filled with material that was T2W-hyperintense and T1W-hypointense (right side) and T2W-hypointense and T1W-isointense (left side). The material in the left tympanic cavity extended into the horizontal ear canal. Bilaterally, the lining of the tympanic cavities was irregularly thickened and contrast enhancing with a nodular appearance on the right. The right tympanic bulla also was thickened. There was a 9.5 mm × 3.5 mm × 11 mm, ovoid mass located in the right nasopharynx that was T2W- and T1W-hyperintense and displayed strong homogeneous contrast enhancement. The medial retropharyngeal lymph nodes were enlarged. Surrounding the external surface of the right tympanic bulla was T2W-hyperintense, strongly contrast-enhancing tissue that extended between the digastricus and longus capitis muscles and into the region of the opening of the tympano-occipital fissure. The MRI finding involving the fascial planes, tissue surrounding the right tympanic bulla, and the tympano-occipital fissure likely represented cellulitis.

Cytologically, the material from the left ear contained 100 cocci per ×100 objective lens and the material from the right ear did not contain any observable microbes. Material from both tympanic cavities was submitted for aerobic culture.

The cat recovered from anesthesia and was discharged on clindamycin[j] (12.5 mg/kg orally *q* 12 hr) while pending culture results. Two days later, the aerobic culture yielded growth of *Staphylococcus felis* from the left ear and *Pasteurella multocida* from the right ear. Based on the sensitivity results, the antibiotics were changed to pradofloxacin[k] (6.9 mg/kg orally *q* 24 hr). The cat was re-evaluated at 2 and 4 wk following discharge. At both evaluations, the owner reported that the episodes of coughing and gagging had resolved. The physical examination was normal without evidence of otitis externa. Neurological examination was normal other than persistent Horner syndrome OD. Based on the improvement, antibiotic therapy was continued for 8 wk.

At 2 mo following discharge, no episodes of coughing or gagging had occurred. Physical and neurological examination were normal with the exception of mild miosis OD. The cat was sedated for a

laryngeal exam using butorphanol[f] (0.27 mg/kg intramuscularly) and alfaxalone[l] (1.9 mg/kg intramuscularly). The previously observed right-sided laryngeal paralysis had resolved. Following laryngeal examination, the cat was induced with propofol[g] (4.4 mg/kg IV) for MRI of the head. The same sequences acquired for the first MRI were repeated along with a dorsal T2W sequence using fat suppression of the neck. On MRI, both tympanic cavities remained filled with material but the lining of the tympanic cavities displayed less contrast enhancement. The contrast-enhancing tissue surrounding the external surface and dissection along tissue planes was no longer present. Images of the neck were normal.

Given the persistent material in the tympanic cavities and miosis OD, antibiotic therapy was continued for an additional 2 mo. At 4 mo following discharge, physical and neurological examination were normal. Antimicrobial therapy was discontinued. One month later, the cat underwent a comprehensive oral health assessment and treatment. At that time, there was no evidence of otitis externa, Horner syndrome OD, or laryngeal dysfunction.

A 7 yr old (5 kg) neutered male domestic shorthair (cat 2) was presented for a 4 day history of lethargy and episodic falling to the left. The owner also noted harsher vocalizations than was normal for the cat. Intermittently, the cat would make exaggerated swallowing attempts independent of eating or drinking.

Physical examination performed by the referring veterinarian was normal. Complete blood count revealed a neutrophilic leukocytosis (white blood cell count 24.98 $\times$ 10^3 cells/μL; reference range 5.50–19.50 $\times$ 10^3 cells/μL and neutrophil count 21.54 $\times$ 10^3 cells/μL; reference range 2.50–14.00 $\times$ 10^3 cells/μL). Serum biochemistry revealed hyperglycemia (285 mg/dL; reference range 77–153 mg/dL), decreased blood urea nitrogen (13 mg/dL; reference range 16–33 mg/dL), hyperproteinemia (9.1 g/dL; reference range 5.2–8.2 g/dL), and hyperglobulinemia (5.5 g/dL; reference range 2.8–4.8 g/dL). The cat received subcutaneous fluids (quantity unknown) and was discharged with instructions to monitor clinical signs. After 4 days, the signs persisted and the cat was referred to the Neurology service at the Veterinary Teaching Hospital, University of Georgia, for further evaluation. The cat had no prior medical history including ear disease, was negative for feline leukemia virus and feline immunodeficiency virus, and was up to date on vaccinations.

On presentation, the physical examination, including otoscopic evaluation, was normal. Neurologically, the cat had normal mentation, gait, postural reactions, and spinal reflexes. CNs were normal with the exception of a left head tilt, rotatory nystagmus with fast phase directed to the right, and miosis in the left eye. Neurological signs were consistent with a lesion affecting the left peripheral vestibular system and miosis in the left eye likely due to partial loss of sympathetic innervation to the eye. Three-view thoracic radiographs were normal. The cat was anesthetized for an MRI of the head. Immediately prior, the cat was sedated with butorphanol[f] (0.2 mg/kg IV) and propofol[g] (4 mg/kg IV but given to the effect of sedation that enabled visualization of the larynx without causing apnea). Laryngeal examination revealed left-sided laryngeal paralysis.

MRI of the head and neck as far caudal as the T3 vertebra was performed. The same multiplanar images as were acquired in cat 1 were acquired in cat 2. Additionally, fat-suppressed dorsal T2W and postcontrast T1W images were acquired.

On MRI, the left tympanic cavity was filled with T2W-hyperintense material within thickening of the tympanic bulla and contrast enhancement of the tympanic cavity lining. The left medial retropharyngeal lymph node was enlarged. Similar to cat 1, the tissues surrounding the external surface of the left tympanic bulla were enlarged, T2W-hyperintense, T1W-isointense, and strongly contrast enhancing. The contrast-enhancing tissue surrounding the left tympanic bulla extended into the region of the tympano-occipital fissure and also extended caudally along the course of the vagosympathetic trunk and common carotid artery to the level of the C2 vertebra. No other abnormalities were observed in the neck. The MRI findings were consistent with left-sided OMI with cellulitis.

Following recovery from anesthesia, the cat was discharged on amoxicillin-clavulanic acid[m] (12.5 mg/kg *per os q* 12 hr) for 6 wk. Twenty days later, physical examination remained normal and all neurological signs had resolved. The owner reported return of normal vocalization and resolution of exaggerated swallowing attempts. Based on the resolution of neurological signs, the cat was discharged with instructions to complete the previously prescribed antibiotic therapy.

Discussion

In both cats, the MRI findings were consistent with OMI, including material in the tympanic cavity with variable signal intensities, tympanic bulla thickening, and contrast enhancement of the lining of the tympanic cavity.[11,12] In addition, both cats had extensive cellulitis of the tissues surrounding the tympanic bulla that extended into the tympano-occipital fissure.

Typically, neurological deficits associated with OMI include dysfunction of the facial nerve (CN VII) and the vestibulocochlear nerve (CN VIII) and Horner syndrome.[13] The facial and vestibulocochlear nerves emerge adjacent to each other from the medulla to exit the cranial cavity via the internal acoustic meatus.[14] Once inside the internal acoustic meatus, the facial nerve travels via the facial canal within the petrosal bone to exit the skull via the stylomastoid foramen.[14] Along its course within the facial canal, the facial nerve is exposed to the tympanic cavity, where it is susceptible to injury.[15] Dysfunction of the facial nerve causes facial paresis/paralysis,

resulting in lagophthalmos, inability to move the pinna, and a lip droop.[15] The receptors for the vestibulocochlear nerve reside in the inner ear and provide for vestibular function and hearing. Dysfunction of the vestibulocochlear nerve results in a head tilt, abnormal nystagmus, positional strabismus, vestibular ataxia, and hearing loss.[16] Finally, the exact course the sympathetic axons take from the cranial cervical ganglion located medial to the tympanic bulla to the eye remains debated. Similar to the facial nerve, the postganglionic sympathetic axons are likely exposed to the tympanic cavity.[17] Loss of sympathetic innervation results in Horner syndrome. The courses of the facial, vestibulocochlear, and sympathetic axons relative to the middle and inner ears explain the development of the observed neurological deficits associated with OMI. Although neither had signs of facial paresis/paralysis, OMI resulted in vestibular dysfunction and/or Horner syndrome in the cats described here.

Laryngeal paralysis suggests dysfunction of the vagus (CN X) or its branch, the recurrent laryngeal nerve. The vagus nerve provide innervation to the striated muscles of the pharynx, larynx, and esophagus. The vagus nerve emerges from the medulla to exit the cranial cavity via the jugular foramen and ultimately exit from the skull via the tympano-occipital fissure to course caudally as the vagosympathetic trunk.[14] The recurrent laryngeal nerve leaves the vagus in the cranial mediastinum and courses cranial along the trachea to innervate the abductors of the larynx.[14] As the tympano-occipital fissure is located caudomedial to the tympanic bulla, the cellulitis that extended into the opening of the tympano-occipital fissure on MRI likely affected the vagus nerve in both cases. Other pathological processes that may cause gagging, coughing, and exaggerated swallowing attempts include glossopharyngeal nerve dysfunction or structural disease affecting the oropharynx, larynx, vagosympathetic trunk, or recurrent laryngeal nerves in the neck. Both cats had normal gag reflexes and, with the exception of a nasopharyngeal polyp in cat 1, had normal cross-sectional imaging of the oropharynx, larynx, and neck. However, had the nasopharyngeal polyp been the cause of the clinical signs, it seems unlikely that signs would have resolved without excision of the polyp. Therefore, the clinical signs were attributed to laryngeal paralysis secondary to extensive cellulitis leading to dysfunction of the vagus nerve. In further support, antibiotic therapy in both cats resolved the clinical signs of laryngeal dysfunction in both cats. In cat 1, laryngeal function was restored concurrent with resolution of the cellulitis as observed on repeat MRI.

Laryngeal paralysis remains uncommon in cats. However, it is possible that laryngeal dysfunction may go unrecognized in cats with middle ear disease that display dysphagia or dysphonia because alternative explanations such as inflammatory polyps or pharyngeal swelling and inflammation often exist concurrently. In the two cats herein, the gagging, coughing, dysphonia, and exaggerated swallowing prompted evaluation of laryngeal function.

Clear evidence-based guidelines for consideration of surgical intervention for the treatment of OMI in cats are lacking. Despite this, surgical therapy for OMI in cats may be considered in those with chronic or recurrent OMI, those with severe neurological deficits, and those failing to respond to medical therapy. In the present cases, medical therapy was pursued as both cats had an acute onset of signs and no antecedent history of otitis and, with the exception of the laryngeal paralysis, the neurological deficits were considered mild. An important implication of identifying laryngeal paralysis in cats with OMI relates to treatment by ventral bulla osteotomy (VBO). In a study of 282 cats undergoing VBO, postoperative respiratory complications were experienced in 9–47% of cats treated with unilateral, staged bilateral, or single-stage bilateral VBO, and 32% of cats with severe postoperative respiratory complications died or were euthanized.[18] Interestingly, dysphagia (4.3%) and dysphonia (2.8%) were reported preoperatively.[18] Although laryngeal dysfunction was not detailed in the report, preoperative respiratory signs were a significant factor for the development of postoperative respiratory complications, highlighting the importance of identifying laryngeal paralysis preoperatively.[18]

Conclusion

Laryngeal paralysis was identified in two cats with OMI and extensive cellulitis surrounding the tympanic bulla and the tympano-occipital fissure. Assessment of laryngeal function should be performed in cats with OMI that present with coughing, gagging, dysphagia, and/or dysphonia. Concurrent laryngeal paralysis may have an impact on the development of postoperative respiratory morbidity and mortality following VBO. In both cats herein, antibiotic therapy resulted in resolution of clinical signs and improved laryngeal function based on visual assessment of the larynx in cat 1. Therefore, in the rare event of laryngeal paralysis secondary to OMI, antibiotic therapy may improve laryngeal function before VBO.

FOOTNOTES

[a] Onsior; Elanco Animal Health, Greenfield, Indiana
[b] Ketaset; Fort Dodge Animal Health, Fort Dodge, Iowa
[c] Midazolam hydrochloride injection; Hospira Inc., Lake Forest, Illinois
[d] Somatom Sensation; Siemens, Erlangen, Germany
[e] Omnipaque 350 mg/mL; GE Healthcare, Princeton, New Jersey
[f] Torbugesic; Zoetis, Kalamazoo, Michigan
[g] Propoflo; Zoetis, Kalamazoo, Michigan
[h] Siemens Skyra 3.0T; Siemens Medical Solutions, Malvern, Pennsylvania
[i] Magnivest; Bayer Healthcare Pharm., Whippany, New Jersey
[j] Antirobe; Zoetis, Kalamazoo, Michigan
[k] Veraflox; Bayer, Shawnee Mission, Kansas
[l] Alfaxalone; Jurox Animal Health, North Kansas City, Missouri
[m] Clavamox; Zoetis, Kalamazoo, Michigan

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