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Emily A Hartman, Robert J McCarthy and Mary A Labato

Abstract

Case summary A 10-year-old neutered male domestic shorthair cat was evaluated because of signs of stertorous breathing and reverse sneezing of 8 months' duration. A CT scan performed 1 week before evaluation indicated nasopharyngeal stenosis or collapse. Increased respiratory effort, stertorous breathing, coughing, reverse sneezing, bilateral black ocular discharge and mucoid left nasal discharge were noted. Rhinoscopy suggested possible nasopharyngeal stenosis. Balloon dilation was attempted but unsuccessful. Ventral rhinotomy was performed the following day using a spring-loaded mouth gag to access the surgical site. After rhinotomy, the patient had neurologic signs attributed to global cerebral ischemia that progressed to respiratory arrest, subsequently resulting in euthanasia.

Relevance and novel information While ischemic brain injury has been associated with the use of a spring-loaded mouth gag in cats, to our knowledge this is the first reported instance where use resulted in respiratory arrest culminating in euthanasia.

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Case description

A 6.0 kg (13.3 lb) 10-year-old neutered male domestic shorthair cat was evaluated for stertorous breathing and reverse sneezing of 8 months' duration. The patient had been treated repeatedly with cefovecin sodium (8.0 mg/kg [3.6 mg/lb] SC) and ciprofloxacin 0.3% ophthalmic drops (1 drop in each nostril, q12h) with no resolution of clinical signs. Eight days before referral, a CT scan of the skull revealed narrowing of the nasopharyngeal lumen without evidence of a mass, suggesting either nasopharyngeal stenosis or collapse. In addition, there was fluid accumulation in the nasal cavity, interturbinate spaces and right frontal sinus. Prednisolone (0.6 mg/kg [0.27 mg/lb] PO q12h) was started at that time, with no signs of improvement.

On presentation, the patient displayed stertorous breathing and reverse sneezing, consistent with the reported history. In addition, there was mild increased respiratory effort, cough, bilateral black ocular discharge and mucoid left nasal discharge. A grade I/VI right-sided

heart murmur was determined by echocardiogram to be clinically insignificant. A complete blood count and blood chemistry panel were both within normal limits.

Rhinoscopy was performed the following day. Oxymorphone (0.1 mg/kg [0.04 mg/lb] IV) and midazolam (0.2 mg/kg [0.09 mg/lb] IV) were administered before the procedure and the patient was induced with propofol (1.8 mg/kg [0.81 mg/lb] IV). Following intubation, the vaporizer was set at 2% isoflurane with a semi-closed rebreathing circuit. A 10 cm spring-loaded mouth

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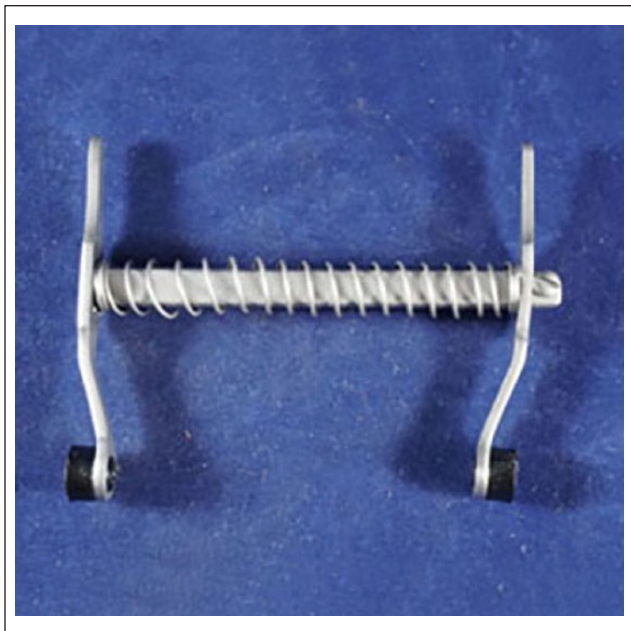


Figure 1 Feline mouth gag

gag (Figure 1) was used to aid placement of the endoscope and maintain visualization. In the retroflexed position rhinoscopy indicated a suspected area of abnormal tissue and mild stenosis in the nasopharynx. Balloon dilation was performed; however, the degree of expansion was considered inadequate. Intraoperatively, the patient maintained a mean arterial pressure between 65 and 80 mmHg. All vital parameters (temperature, heart rate, respiratory rate), pulse oximetry and end-tidal CO₂ were recorded within normal limits throughout the duration of the 40 min anesthesia period. After the rhinoscopy and balloon dilation procedure, one dose of dexamethasone sodium phosphate (0.13 mg/kg [0.05 mg/lb] IV) was administered. Recovery was uneventful with an initial improvement in breathing and decrease of respiratory sounds. Because it was suspected that abnormal tissue remained in the nasopharynx, surgical debridement with excision and biopsy was recommended either in the future if the clinical signs worsened, or at the present time. The owner elected to proceed with surgical intervention the following day.

Ventral rhinotomy was performed the next day. Oxymorphone (0.1 mg/kg [0.04 mg/lb] IV) and midazolam (0.1 mg/kg [0.04 mg/lb] IV) were administered prior to the procedure and the patient was induced with propofol (1.8 mg/kg [0.81 mg/lb] IV). Following intubation, the vaporizer was set at 1.5% isoflurane with a semi-closed rebreathing circuit. The surgery site was again made accessible with the use of a 10 cm spring-loaded mouth gag (Figure 1). Aside from a small amount of mucopurulent discharge from the nasal cavity, no obstructive abnormalities were noted in the

nasopharynx or caudal nasal cavity, although the tissue was soft, friable and reddened. Biopsies were obtained. The patient remained normotensive and all vital parameters (heart rate, respiratory rate, temperature), pulse oximetry, end-tidal CO₂ were within normal limits throughout the 25 min procedure. At the conclusion of surgery, single doses of cefazolin (140 mg [23 mg/kg] IV) and dexamethasone (0.8 mg [0.13 mg/kg] IV) were administered. Upon anesthetic recovery, the patient displayed immediate, marked neurologic abnormalities, including leg paddling and extensor rigidity. Pupillary light reflexes were intact, but there was no apparent menace response, or physiologic nystagmus. The patient was blind. Neurologic examination was consistent with cortical damage, likely secondary to global cerebral ischemia. Four hours after surgery, a venous critical care panel showed no evidence of perfusion inadequacy. Lactate measured 1.6 mmol/l, blood urea nitrogen 28 mg/dl and creatinine 1.2 mg/dl.

Over the following 36 h medical stabilization was attempted. Lactated Ringer's solution with 20 mEq/l KCl was administered IV (1.7 ml/kg/h [0.8 ml/lb/h]) and the patient received buprenorphine (0.005 mg/kg [0.002 mg/lb] IV q6h). Six and 13 h after surgery, seizure activity was noted and treated with diazepam (0.42 mg/kg [0.19 mg/lb] IV). Sixteen hours after surgery, levetiracetam (20 mg/kg [9.07 mg/lb] IV q8h) was instituted for seizure control. Twenty-eight hours after surgery, the venous critical care panel was repeated and showed a lactate of 1.6 mmol/l, blood urea nitrogen of 14 mg/dl and creatinine 1.0 mg/dl, with no indication of perfusion compromise.

No urine production was noted for 18 h after surgery, so two doses of furosemide (2.0 mg/kg [0.91 mg/lb] IV q4h) were given. Shortly after therapy, the patient passed a large amount of urine, but remained dull and blind. Thirty-two hours after surgery acute respiratory arrest occurred. Immediate intubation was performed and resuscitation with positive pressure ventilation was successful. Thoracic radiographs indicated patchy alveolar to interstitial patterns with air bronchograms, most consistent with acute-onset non-cardiogenic pulmonary edema. A venous critical care panel was again repeated which showed marked elevation in lactate (6.0 mmol/l), blood urea nitrogen (15 mg/dl) and creatinine (1.2 mg/dl). Two more seizure events were controlled with diazepam (0.42 mg/kg [0.19 mg/lb] IV), but the patient subsequently experienced a second episode of respiratory arrest and was placed on a ventilator. The owners elected humane euthanasia at that time.

Discussion

Use of a spring-loaded mouth gag has been reported previously to cause neurologic impairment in cats.¹⁻⁷ In a study detailing the problem in 20 cats, all were blind

upon recovery from anesthesia, whereas some demonstrated deafness, head tilt, weakness, opisthotonus, decreased conscious proprioception and central depression. Seventy percent of cats regained vision and returned to normal neurologic status within 14 days, whereas long-term blindness and impairments were recorded in the remaining patients.¹ There was no apparent association between the length of time the mouth was opened and resulting impairment.^{1,2} No associated respiratory compromise was documented.¹

The cause of neurologic impairment in these cats has been elucidated. While in some species (dogs, humans) the main cerebral blood supply arises from both the internal carotid and basilar arteries, cats rely solely on the maxillary branch of the external carotid artery (MB-ECA) to feed the cerebral vascular circle.²⁻⁵ This branch lies in immediate proximity to the caudal aspect of the jaw, and is compressed between the bony tympanic bulla and angular process of the mandible when the mouth is opened to maximal extent.^{3,4,6,7} In addition, the medial and lateral pterygoid muscles and temporalis muscle border the vasculature and exert pressure when the mouth is placed in full extension, compromising flow.^{1,2,3,7} Occlusion of the MB-ECA can cause cerebral hypoxia, resulting in global cerebral ischemia.¹⁻⁷ Further compounding ischemia, the use of anesthesia may cause a reduction in perfusion to vital structures.^{1,2,3,4,6} Areas of the brain most vulnerable to ischemic damage are the cerebral cortex, hippocampus, caudal colliculi and thalamic nuclei.¹

While maximal opening of the mouth in cats is often associated with MB-ECA flow alterations, clinical sequelae are less common. In six cats the amount of force applied to open the mouth was directly correlated with both abnormal waveforms on electroretinography (ERG) and changes on magnetic resonance angiography, but no neurologic abnormalities were noted upon recovery.^{2,3,7} In a second study, 3/6 cats had abnormal brain auditory-evoked responses and ERG waveforms, but none showed abnormal neurologic signs.⁴

Conclusions

To our knowledge this is the first reported case to suggest that the repeated use of a spring-loaded mouth gag may cause respiratory arrest secondarily to presumed intraoperative cerebral ischemia. Although the cat in the present report appeared neurologically normal

following the first procedure, it is possible that some level of cerebral hypoxic injury had occurred, and with reintroduction of the mouth gag on the following day greater damage was sustained. Previous reports are limited to transient deficiencies generally related to forebrain injury and no studies have investigated the repeated use of such devices in a short period of time. Although a necropsy of this patient was not performed to confirm hypoxic injury in the hindbrain causing the ultimate respiratory arrest, recommendations based on the details of this case would be to avoid the use of spring-loaded mouth gags in cats altogether. If maximal opening of the mouth is required for prolonged periods in feline patients, it would be recommended to periodically return the mouth to a closed position to allow perfusion of vital structures and protect from ischemic events.

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