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AMERICAN COLLEGE OF VETERINARY ANESTHESIA AND ANALGESIA

Anesthesia Case of the Month

History

A 4-month-old 1.6-kg (3.5-lb) sexually intact female domestic shorthair kitten was referred to the North Carolina State University Small Animal Emergency Service because of progressive respiratory distress. One week earlier, the referring veterinarian had sedated the kitten with IM administration of dexmedetomidine, ketamine, and butorphanol (doses unknown) for radiographic examination because of a 1.5-month history of nasal discharge from the left nostril combined with increased respiratory rate and effort that had not responded to medical management. While sedated, the kitten became cyanotic. Intubation attempts failed because of an undefined oropharyngeal obstruction; thus, supplemental O_2 was provided by face mask, and atipamezole (dose unknown) was administered IM. The kitten recovered but had progressive dyspnea over the following week, which prompted the present referral. A single lateral radiographic image (not shown) had been obtained by the referring veterinarian, and findings included hyperinflated lungs, diffuse megaesophagus, and a gas-filled gastrointestinal tract.

On referral examination, the kitten was alert and responsive yet dyspneic with a respiratory rate of 30 to 40 breaths/min (reference range, 16 to 40 breaths/min), increased respiratory effort, stertor, and dynamic outward excursions of the thoracic inlet during inspiration. The kitten had a heart rate of 200 beats/min (reference range, 150 to 200 beats/min), capillary refill time < 2 seconds with pink mucous membranes, rectal temperature of 36.5°C (97.7°F; reference range, 37.8°C to 39.5°C [100°F to 103.1°F]), and no history or evidence of trauma. With handling, the kitten's respiratory effort markedly worsened to open-mouth breathing. A 22-gauge IV catheter was aseptically placed in a cephalic vein, blood was collected for basic analyses, and the kitten was placed in an O_2 cage set at 40%. Hematologic analyses revealed that the kitten had a PCV of 44% (reference range, 30% to 45%), plasma concentration of total solids of 8.8 g/dL (reference range, 5.7 to 8.0 g/dL), BUN concentration[a] of 5 to 15 mg/dL (reference range, 10 to 30 mg/dL), and blood glucose concentration[b] of 120 mg/dL (reference range, 63 to 132 mg/dL). Further diagnostic procedures were postponed because of the kitten's respiratory instability, and the kitten remained in the O_2 cage and was monitored overnight.

The kitten's respiratory signs worsened overnight, and by morning, the kitten was quiet, dyspneic, and intermittently open-mouth breathing. Our primary differential diagnosis list for progressive upper airway obstruction in a young cat included nasopharyngeal polyps, nasopharyngeal stenosis, congenital anatomic abnormalities, and upper airway congestion secondary to infection. Given the progressive and long-term nature of the condition, the presence of outward excursions during respiration, the age of the kitten, and the referring veterinarian's reported inability to intubate the kitten, a feline nasopharyngeal polyp (FNP) was suspected. Thus, CT of the kitten's head followed by nasopharyngeal and laryngeal endoscopic evaluations was recommended.

Prior to anesthetic induction, we prepared for a likely difficult intubation and ensured functionality of all readied equipment, including multiple sizes of endotracheal tubes (ETTs), a long flexible stylet to assist in ETT placement, a laryngoscope, a capnography unit, and supplies for suction, temporary tracheostomy, and stylet-guided retrograde intubation along with Kelly and Allis tissue forceps for the potential removal of a nasopharyngeal polyp to allow for orotracheal intubation. The kitten was transported to the anesthesia area in a transparent induction chamber connected to an anesthetic circuit delivering 100% O_2 (5 L/min). When the kitten was removed from the chamber, supplemental 100% O_2 (3 L/min) was provided by face mask, and a reflectance pulse oximetry probe[c] was placed on the ventral aspect of the tail. The O_2 saturation of hemoglobin measured with pulse oximetry (Spo_2) was 87% (reference range, 95% to 100%).

No premedications were administered owing to concerns for the kitten's fragile respiratory status and the potential effects of such drugs worsening the kitten's airway obstruction. Anesthesia was rapidly induced with etomidate (4.3 mg/kg [1.95 mg/lb], IV, to effect) while the kitten was simultaneously equipped for continuous 3-lead ECG monitoring with a multiparameter monitor,[d] and a Doppler ultrasonic flow detector[e] was placed over the right plantar artery.

Orotracheal intubation was unsuccessful because of a large, firm mass in the nasopharynx. The mass completely obstructed laryngeal visualization, was presumed to have been an FNP, and was quickly removed by manual traction with Kelly forceps **(Figure 1)**. Subsequent hemorrhage in the oropharyngeal area was suctioned,[f] the arytenoid cartilages were visualized, and then the kitten was

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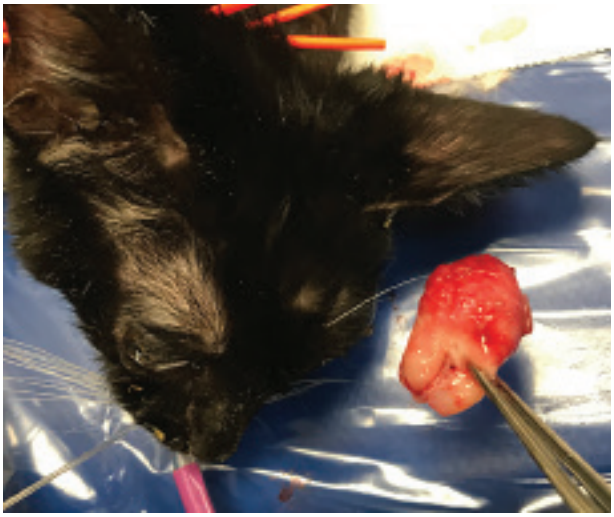

Figure 1—Image of an anesthetized and intubated 4-month-old 1.6-kg (3.52-lb) sexually intact female domestic shorthair kitten with a 1.5-month history of dyspnea and unilateral nasal discharge that were nonresponsive to treatment alongside the nasopharyngeal polyp (approx 3.0 × 1.8 × 1.6 cm) removed by manual traction from the kitten's oropharynx during the present anesthetic event. The polyp is held near the kitten's head to underscore the relatively large size of the polyp, compared with the size of the kitten.

intubated with a 3-mm-internal-diameter cuffed Murphy-type ETT.[g] Following intubation, the kitten was apneic and had a pulse rate of 210 beats/min (measured with a Doppler ultrasonic flow detector). Capnography and manual positive-pressure ventilation (100% O_2; peak inspiratory pressure, < 20 cm H_2O) were initiated. No end-tidal CO_2 concentration ($ETco_2$) or waveform was detected on capnography, and slightly increased resistance to manual ventilation was noticed by the anesthesiologist (LFG). An airway occlusion was suspected. The ETT was removed for inspection, and nothing abnormal about it was detected. Regardless, a new ETT of the same size was used for orotracheal intubation. Proper ETT placement was confirmed with visualization of the ETT passing between the arytenoid cartilages. The $ETco_2$ remained at 0 and no waveform was detected on capnography. The kitten's heart rate decreased to 160 beats/min, with heart sounds and peripheral pulses detected with thoracic auscultation and the Doppler ultrasonic flow detector, respectively. The kitten had decreased bronchovesicular sounds, and the Spo_2 remained 87%.

Question

What were possible causes for no detected $ETco_2$ or waveform on capnography combined with low Spo_2 for this kitten?

Answer

Differential diagnoses for the absence of $ETco_2$ after successful intubation include capnography malfunction, cardiopulmonary arrest causing a complete lack of circulation delivering CO_2 to the alveoli, apnea with the subsequent absence of CO_2 transport from the alveoli to the capnographic sensor, and a complete airway obstruction preventing CO_2 exhalation. To evaluate equipment function, the side-stream capnography adaptor was removed from the ETT, and the anesthesiologist (LFG) blew into the sensor without making physical contact with the adaptor. An $ETco_2$ and concurrent waveform registered on the monitor, reconfirming proper equipment function. Simultaneously, the presence of a heartbeat and pulse was confirmed by thoracic auscultation and the Doppler ultrasonic flow detector, respectively. Because myocardial contraction and forward blood flow should deliver CO_2 to the lungs, the absence of $ETco_2$ was not attributable to cardiac arrest. Although the kitten was apneic, intermittent positive-pressure ventilation was immediately implemented following intubation; thus, apnea was also not responsible for the observed issue. The slight resistance to manual ventilation and the bilaterally decreased bronchovesicular sounds prioritized the likelihood of an airway obstruction.

The ETT was suctioned,[h] and an approximately 5-cm-long blood clot **(Figure 2)** was retrieved from the lumen of the ETT. Immediately, the kitten had substantially improved bronchovesicular sounds in all lung fields and more prominent chest excursions. The resistance to manual ventilation resolved, Spo_2 climbed to 100%, and capnography waveforms and $ETco_2$ were detected. The kitten's initially recorded $ETco_2$ was 90 mm Hg (reference range, 30 to 40; measurement range of the capnography equipment,[1] 0 to 99 mm Hg), which further supported relative hypoventilation secondary to airway obstruction.

Manual positive-pressure ventilation was continued to achieve an $ETco_2$ between 30 and 40 mm Hg. The kitten remained stable with a continued Spo_2 of 100%, and an oropharyngeal examination revealed no further abnormalities. Thus, our presumptive diagnosis was FNP, and CT of the kitten's head followed by nasopharyngeal and laryngeal endoscopic evaluations was canceled pending histologic examination of the removed tissue. The kitten recovered from anesthesia smoothly and was extubated without difficulty. Histologic results were most consistent with a benign inflammatory polyp, such as FNP.

The kitten was presented 2 months later with less severe clinical signs that included mild nasal discharge and sneezing but no overt dyspnea. Supplemental O_2 was not needed, and hematologic results were within reference limits. The kitten underwent general anesthesia, including routine orotracheal intubation, without complication, for CT of its head. Findings on CT indicated a nasopharyngeal polyp on the left side, and the polyp was successfully removed with manual traction. Recovery and hospital discharge were unremarkable, and no further recurrence was reported.

Figure 2—Image of the approximately 5-cm-long blood clot that occluded the lumen of the endotracheal tube following removal of the polyp from the kitten described in Figure 1. For size reference, the clot is on a 10.16 X 10.16-cm (4 X 4-inch) gauze sponge.

Discussion

Feline nasopharyngeal polyps, also known as feline inflammatory polyps, pharyngeal polyps, aural polyps, and middle ear polyps,[2–6] have unknown etiology and usually occur in younger cats, typically < 2 years of age. Clinical signs can range from mild nasal discharge, sneezing, and vocal change to marked dyspnea.[2] In the kitten of the present report, the large polyp (approx 3.0 X 1.8 X 1.6 cm) relative to this kitten's small size caused almost complete pharyngeal obstruction and prevented routine orotracheal intubation. Generally, diagnostic procedures for FNP may include oropharyngeal examination, radiography, rhinoscopy, and CT.[2] Although manual traction can be effective in FNP removal and subsequent relief of a compromised airway, the approach typically does not remove the base of the mass, and recurrence is possible.[2,4,6] Additionally, a perceived lack of improvement or the need for additional intervention may occur with bilateral FNP formation. Surgical removal by manual traction, combined with ventral bulla osteotomy, may be recommended to reduce the potential of regrowth.[2,6] With FNP removal by manual traction alone, anti-inflammatory treatment is often recommended to reduce the risk of recurrence.

In the kitten of the present report, manual removal of the presumed FNP resulted in localized hemorrhage that entered the trachea and caused a blood clot that obstructed the lumen of the ETT. Other causes for ETT obstruction include excessive airway secretions, additional obstructive abnormalities (eg, tracheal mass or tracheal collapse), or malpositioning or a physical kink of the ETT.[7]

Hypercapnia ($ETCO_2$ > 40 mm Hg or partial pressure of CO_2 dissolved in plasma > 45 mm Hg), also known as hypercarbia, is the hallmark of hypoventilation. With airway obstructions, especially those within the ETT, hypercapnia can be substantial.[7] Because the diffusion rate of CO_2 across alveolar-capillary membranes is approximately 20 times that of O_2,[8] resolution of ETT obstruction resulted in swift normalization of this kitten's $ETCO_2$.

Severe hypoxemia is defined as an SpO_2 < 90%.[9] This kitten's degree of hypoxemia (SpO_2, 87%) was consistent with acute airway obstruction, and administration of supplemental O_2 likely minimized further decline in SpO_2. Relief of the obstruction also allowed rapid correction of hypoxemia.

To confirm hypoxemia and the degree of hypercapnia, it would have been ideal to have sampled arterial blood to evaluate the arterial O_2 saturation of hemoglobin and the partial pressures of O_2 and CO_2. However, as the team's full focus was on mitigating the urgent airway obstruction, these evaluations were not performed.

Etomidate is a rapid-acting, nonbarbiturate anesthetic induction agent. The major advantage of etomidate, compared with other induction agents, is that it results in minimal cardiopulmonary depression.[10,11] From our clinical experience, patients with substantial airway obstruction maintain ventilation during induction with etomidate. Furthermore, a study[12] shows no significant decrease in tidal volume or change in minute volume in dogs administered etomidate at doses up to 3.0 mg/kg (1.36 mg/lb) IV. Because of the kitten's level of respiratory compromise, decline during overnight hospitalization, and suspected airway obstruction, we selected etomidate for induction to minimize further respiratory depression and hypoxemia prior to securing a patent airway. The relatively high dose of etomidate needed for successful induction without premedication only transiently suppressed spontaneous ventilation.

Disadvantages of etomidate include high cost, transient adrenocortical inhibition (can occur following a single induction dose in dogs[13]), excitation, myoclonus, signs of pain on injection, and poor recovery quality[14]; however, induction and recovery quality in this kitten were rapid and smooth. Because of the propylene glycol carrier and subsequent high osmolarity of etomidate, hemolysis and hemoglobinuria may occur following etomidate administration in dogs.[15] Similarly, the kitten of the present report had faint red discoloration of urine voided spontaneously during recovery, and this was presumed to have been hemoglobinuria secondary to etomidate; the condition resolved without treatment. To our knowledge, little information regarding etomidate and hemolysis in cats exists in the veterinary literature. Anecdotally, hemoglobinuria has occurred in about one-third of cats treated with etomidate.[i] To confirm whether etomidate caused hemoglobinuria in this kitten, it would have been ideal to have performed a urinalysis and measured a postanesthetic PCV. Unfortunately, the kitten's urine soaked into an absorbent pad, and

the kitten's demeanor combined with its small size prevented successful venipuncture.

In hindsight, despite thorough planning for an anticipated difficult intubation, a more extensive plan for the mitigation of procedural hemorrhage would have been ideal. For instance, to have prevented gravitational draining of blood into the trachea, the patient could have been positioned in a Trendelenburg (head-down) position. Although this would have allowed blood to drain rostrally, it also could have caused increased respiratory compromise from increased pressure on the diaphragm. Additionally, Trendelenburg positioning would have impaired visualization of the oral cavity, which was critical given the circumstances. Although active suction was readily available and quickly used to clear the oropharyngeal area and allow visualization of the arytenoid cartilages, perhaps it could have been used simultaneously to the extent possible during FNP removal. Furthermore, a vasoconstrictive product such as epinephrine (1:200,000) could have been used topically in a manner similar to the use of various vasoconstrictive products in human medicine.[16,17] However, for the kitten of the present report, effective use of a topical vasoconstrictive product would have been challenging owing to the large size of the FNP relative to oropharyngeal diameter. Additionally, attempts at such application would have delayed polyp removal, and we felt it was more important to expedite removal and intubate the kitten.

Multiple factors contributed to the successful outcome of this airway emergency. First, advanced planning meant all necessary equipment was available and ready for use. Second, the team approach enabled several aspects of the case to move quickly and seamlessly (eg, induction simultaneous with monitoring placement), allowing for rapid resolution of the problem. Third, the appropriate choice of induction agent may have minimized the marked desaturation and cyanosis that commonly affect patients with airway emergencies. Fourth, with the use of capnography, we were alerted to the fact that despite visual confirmation of proper ETT placement, the airway still was not patent and warranted further investigation. Fifth, the availability of appropriately sized suction tips allowed us to immediately clear the oropharyngeal area and later the ETT lumen; this action saved the kitten's life. Ultimately, this case illustrated the value of advanced planning and efficient teamwork when treating patients with compromised airways.

Footnotes

a. Azostix, Siemens Healthcare Diagnostics Inc, Tarrytown, NY.
b. AlphaTrak2 blood glucose test, Zoetis Inc, Kalamazoo, Mich.
c. Rad-57 Pulse Oximeter, Masimo Corp, Irvine, Calif.
d. Passport Multiparameter Monitor, Mindray DS US Inc, Mahwah, NJ.
e. Doppler Flow Detector-Model 811-B, Parks Medical Electronics, Aloha, Ore.
f. Airlife Tri-flo 18 French Suction Catheter, Vyaire Medical Inc, Mettawa, Ill.
g. Dee Veterinary Products, Miami Gardens, Fla.
h. MediChoice 6F coiled suction catheter, Owens and Minor, Mechanicsville, Va.
i. Graham L, Associate Clinical Professor of Anesthesiology, College of Veterinary Medicine, North Carolina State University, Raleigh, NC: Personal communication, 2020.

References

1. Shenzhen Mindray Bio-Medical Electronics Co Ltd. Passport 12/Passport 8 patient monitor operator's manual. Available at: www.mindraynorthamerica.com/wp-content/uploads/2020/03/H-046-004417-00-Passport-8_12-Ops-Manual-FDA-13.0.pdf. Accessed Dec 29, 2020.
2. American College of Veterinary Surgeons. Nasopharyngeal polyps page. Available at: www.acvs.org/small-animal/nasopharyngeal-polyps. Accessed Dec 18, 2020.
3. Donnelly KE, Tillson DM. Feline inflammatory polyps and ventral bulla osteotomy. *Compend Contin Educ Pract Vet* 2004;26:446–454.
4. Kapatkin AS, Matthiesen DT. Results of surgery and long-term follow-up in 31 cats with nasopharyngeal polyps. *J Am Anim Hosp Assoc* 1990;26:387–392.
5. Kudnig ST. Nasopharyngeal polyps in cats. *Clin Tech Small Anim Pract* 2002;17:174–177.
6. MacPhail CM, Innocenti C, Kudnig ST, et al. Atypical manifestations of feline inflammatory polyps in three cats. *J Feline Med Surg* 2007;9:219–225.
7. Mosley CA. Anesthesia equipment. In: Grimm KA, Lamont LA, Tranquilli WJ, et al, eds. *Lumb & Jones' veterinary anesthesia and analgesia*. 5th ed. Ames, Iowa: Wiley-Blackwell, 2015;23–85.
8. Stoelting RK, Hillier SC. Pulmonary gas exchange and blood transport of gases. In: *Pharmacology and physiology in anesthetic practice*. 4th ed. New York: Lippincott Williams and Wilkins, 2006;783–793.
9. Haskins SC. Monitoring anesthetized patients. In: Grimm KA, Lamont LA, Tranquilli WJ, et al, eds. *Lumb & Jones' Veterinary anesthesia and analgesia*. 5th ed. Ames, Iowa: Wiley-Blackwell, 2015;86–113.
10. Kissin I, Motomura S, Aultman DF, et al. Inotropic and anesthetic potencies of etomidate and thiopental in dogs. *Anesth Analg* 1983;62:961–965.
11. Rodríguez JM, Muñoz-Rascón P, Navarrete-Calvo R, et al. Comparison of the cardiopulmonary parameters after induction of anaesthesia with alphaxalone or etomidate in dogs. *Vet Anaesth Analg* 2012;39:357–365.
12. Nagel ML, Muir WW, Nguyen K. Comparison of the cardiopulmonary effects of etomidate and thiamylal in dogs. *Am J Vet Res* 1979;40:193–196.
13. Dodam JR, Kruse-Elliott KT, Aucoin DP, et al. Duration of etomidate-induced adrenocortical suppression during surgery in dogs. *Am J Vet Res* 1990;51:786–788.
14. Muir WW III, Mason DE. Side effects of etomidate in dogs. *J Am Vet Med Assoc* 1989;194:1430–1434.
15. Ko JCH. Acute haemolysis associated with etomidate-propylene glycol infusion in dogs. *J Vet Anaesth* 1993;20:92–94.
16. Korkmaz H, Yao WC, Korkmaz M, et al. Safety and efficacy of concentrated topical epinephrine use in endoscopic endonasal surgery. *Int Forum Allergy Rhinol* 2015;5:1118–1123.
17. Khosla AJ, Pernas FG, Maeso PA. Meta-analysis and literature review of techniques to achieve hemostasis in endoscopic sinus surgery. *Int Forum Allergy Rhinol* 2013;3:482–487.