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Severe hypercapnia associated with a chemodectoma causing profound tracheal narrowing in a cat

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TITLE OF CASE <i>Do not include "a case report"</i>
Severe hypercapnia associated with a chemodectoma causing profound tracheal narrowing in a cat
SUMMARY <i>Up to 150 words summarising the case presentation and outcome (this will be freely available online)</i>
An eleven-year-old, domestic short haired cat presented out-of-hours with a one-day history of dyspnoea and open-mouthed breathing. Ninety minutes after arrival the cat acutely deteriorated. Butorphanol (0.2 mg/kg) was administered intravenously via a pre-placed cephalic vein cannula, after which anaesthesia was induced (1.3 mg/kg alfaxalone). The cat's trachea was intubated with a 3.5 mm internal diameter cuffed endotracheal tube (ETT) but it was not possible to give a manual breath and a capnograph trace was absent; therefore the ETT was replaced with a 4.5 mm internal diameter cuffed ETT. A manual breath was still not possible but a capnograph trace was present [end-tidal CO ₂ tension of 95 mmHg (12.7 kPa)]. Intravenous dexamethasone (0.15 mg/kg) and terbutaline (0.01 mg/kg) were administered. Computed tomography revealed a heart base mass compressing the trachea and decreasing its lumen to 1 mm diameter. The cat was euthanised due to poor prognosis. Histopathology revealed a chemodectoma.

BACKGROUND *Why you think this case is important – why did you write it up?*

This report presents a unique case of severe hypercapnia during general anaesthesia (GA) in a cat, secondary to marked tracheal compression and narrowing by a chemodectoma (heart base tumour). It also highlights the importance of adequate airway management, especially when these tumours significantly decrease tracheal diameter in an emergency, out-of-hours scenario.

Chemodectomas are rare tumours in cats with only a few previously reported cases.

Hypoventilation leading to hypercapnia [end tidal CO₂ (ETCO₂) tension > 45 mmHg (6.0 kPa)] is a common complication seen during small animal anaesthesia with a relative frequency of 13.5% seen in all dog and cat general anaesthetics at a referral hospital (1). Although there are many potential causes of hypercapnia during GA, treatment normally consists of ventilating the animals' lungs to increase minute ventilation by increasing respiratory rate and/or tidal volume, thereby reducing ETCO₂ tension (2).

Reductions in airway diameter and the resultant increase in resistance may lead to severe hypercapnia and hypoxaemia and this should be considered when managing these cases.

CASE PRESENTATION *Presenting features, clinical and environmental history*

An eleven-year-old, 4.5 kg, male neutered, domestic short haired cat presented as an out of hours emergency with a one-day history of acute onset progressively worsening dyspnoea. On arrival the cat was open-mouthed breathing and stertorous with markedly increased inspiratory and expiratory effort; respiratory rate was 40 breaths per minute. Heart rate was 180 beats per minute with no audible murmur on auscultation. The American Society of Anesthesiologists status was IV-E. A 22-gauge intravenous (IV) cannula was placed aseptically into the right cephalic vein and the cat was placed into an oxygen kennel in the intensive care unit with, an inspired fraction of oxygen of 50%. Due to the acute presentation, upper airway obstruction was suspected and therefore the initial plan was to perform bronchoscopy during GA, with more advanced imaging if required.

INVESTIGATIONS *If relevant*

The cat became increasingly stressed after hospitalisation. Approximately ninety minutes after arrival, following consultation and initial clinical examination, respiratory distress became significantly worse, probably due to the stress of hospitalisation. The cat received 0.2 mg/kg butorphanol (x) IV which produced mild sedation, but the cat deteriorated further and became cyanotic with hypersalivation. Approximately 5 minutes after butorphanol administration, GA was induced with alfaxalone (x) to effect, at a total dose of 1.3 mg/kg IV. The cat's trachea was intubated with a 3.5 mm internal diameter, polyvinyl chloride (PVC), cuffed ETT (x), which was connected to a Mapleson D T-piece delivering oxygen at 2 litres per minute. No capnograph trace and no thoracic movement were observed after delivery of a manual breath. The position of the ETT was checked and confirmed as correctly positioned within the trachea. A portable pulse oximeter (x) was attached to the cat's tongue which showed a percentage saturation of arterial haemoglobin with oxygen (SPO₂) of 80%. There was a concern that the ETT could be obstructed so it was removed and replaced with a 4.5 mm internal diameter, PVC, cuffed ETT. With the new ETT in place it was still not possible to give an adequate manual breath because the paediatric adjustable pressure-limiting (APL) valve on the breathing system had an opening pressure of approximately 30 cmH₂O when closed it proved impossible to generate pressures high enough to deliver an adequate tidal volume. The cat began breathing spontaneously with a paradoxical breathing pattern and exaggerated effort. A capnograph trace was present with an ETCO₂ tension of 95 mmHg (12.7 kPa). There was no visible obstruction within the ETT that had been removed. The SpO₂ gradually increased as the cat started to breathe spontaneously and remained between 99 - 100% for the entire 40 minutes of GA. General anaesthesia was maintained with isoflurane (mean end-tidal isoflurane of 1.1%) vaporised in oxygen. Monitoring consisted of sidestream gas analysis, electrocardiogram (ECG), non-invasive oscillometric blood pressure (x) and pulse oximetry. Due to the difficulty with manual ventilation terbutaline (x) at a dose of 0.01 mg/kg was administered slowly by IV to produce bronchodilation. Dexamethasone (x) at a dose of 0.15 mg/kg was also administered IV for its potent anti-inflammatory effects. No improvement in lung compliance was noted after administration of either of these drugs.

Five minutes after induction of anaesthesia the cat was moved to diagnostic imaging for a

computed tomography (CT) scan while still attached to a portable anaesthetic machine with the same breathing system and monitoring described above. The cat was breathing spontaneously with a respiratory rate of 9 - 11 breaths per minute but ETCO₂ tension remained high at 98 - 110 mmHg (13.1 - 14.7 kPa) throughout the CT scan. Attempts at manual ventilation were unsuccessful; the APL valve reached its safety opening pressure without achieving any visible expansion of the thorax. The cat also demonstrated a paradoxical breathing pattern during spontaneous ventilation. The CT scan was performed without incident and pre and post contrast (x) images of the head, thorax and abdomen were obtained. A large heart base mass surrounding and compressing the trachea was identified. The mass was compressing the trachea externally with no evidence of direct intraluminal invasion. The compression caused by the mass led to severe narrowing of the tracheal lumen to a width of 1 mm with normal trachea cranial and caudal to the mass (Fig.1). Maximal tracheal compression was seen at the level of the fifth thoracic vertebra. Other CT findings included multifocal consolidation of the lung lobes possibly due to absorption atelectasis or the increased negative intrathoracic pressure generated during inspiration, pulmonary bullae and bilateral rhinitis which may have explained the stertor noted prior to GA. No other significant abnormalities were detected in the remaining organs.

The cat was moved into the surgical preparation area using a portable anaesthetic machine while the CT findings and treatment options were discussed with the owners. By this point ETCO₂ tension was 123 mmHg (16.4 kPa) with a respiratory rate of 9-13 breaths per minute. Other clinical parameters were acceptable throughout GA with an SPO₂ of 99 - 100%, mean arterial pressure of 73 - 90 mmHg, and a rectal temperature of 37.1 °C taken after CT. The poor prognosis was communicated to the owners but surgical exploration and placement of a tracheal stent was offered. Due to the poor prognosis the owners elected to euthanise the cat. Total anaesthetic time was 40 minutes from induction to euthanasia.

DIFFERENTIAL DIAGNOSIS *If relevant*

Hypercapnia was considered to be caused primary disease process but prior to the CT scan five other causes of hypercapnia were considered.

- Excessive anaesthetic depth: excluded by examining the cat's eye position and the end-tidal concentration of isoflurane which was 1.1%.
- Inappropriate fresh gas flow: fresh gas flow was calculated according to the cat's bodyweight, respiratory rate and breathing system; there was no rebreathing of CO₂ which was confirmed by capnography.
- Pneumothorax: auscultation during spontaneous breathing indicated that this was unlikely. Also, CT images were not consistent with pneumothorax.
- The cat's rectal temperature was 37.1 °C at the end of GA so increased production of carbon dioxide due to pyrexia was deemed unlikely.
- Partial airway obstruction: we had already changed the ETT and partial obstruction was excluded; however there was still the possibility of a foreign body or a mass within the trachea.

TREATMENT *If relevant*

Treatment of hypercapnia would have been by IPPV using a mechanical ventilator as attempts at manual ventilation were unsuccessful. Use of a mechanical ventilator would have eliminated the paediatric APL valve on the Mapleson D breathing system, thus allowing higher inspiratory pressures to be generated. When the acute deterioration occurred airway patency was the main priority and was managed by placing an ETT of an adequate size, attaching monitoring equipment and planning an optimal CT protocol to reach a diagnosis. In retrospect a portable ventilator might have helped to manage the hypercapnia.

No further treatment was provided once imaging had been performed and the cat was euthanised due to the poor prognosis. If surgery had proceeded a mechanical ventilator was available in the main theatre.

OUTCOME AND FOLLOW-UP

A post-mortem examination was performed and revealed an expansile, solid mass at the base of the heart which was infiltrating around the great vessels and compressing the trachea. A nodule was also noted at the ventral margin of the left caudal lung lobe. Histopathology showed that the heart base mass was a chemodectoma most likely arising from the aortic body. The lung mass was believed to be a low-grade pulmonary adenocarcinoma; however, immunohistochemistry was not performed and therefore metastasis from the chemodectoma could not be excluded.

DISCUSSION *Include a very brief review of similar published cases*

This report describes a case of severe tracheal narrowing caused by mechanical compression by a chemodectoma leading to acute dyspnoea with resultant hypoventilation and hypercapnia. Furthermore, it describes the potential complications that might be anticipated when these cases require GA.

Cardiac tumours in companion animals are rare with a reported incidence of 0.0275% in cats (3). Chemodectomas are neuroendocrine tumours of chemoreceptor cells that can be found

in either the carotid or aortic bodies located at the base of the heart (3). Cats with chemodectomas present with dyspnoea of variable origin including pulmonary oedema (4), lymphocyte rich pleural effusion (5) and uncharacterised pleural effusion (6). Long term prognosis is poor with survival ranging from immediate euthanasia (4) to 19 months. There is little information about the treatment of chemodectomas in cats, although treatment options in dogs have been reported (7,8). Toceranib phosphate as a treatment for inoperable heart base tumours led to improved clinical signs despite minimal changes in tumour size in 19 out of 21 dogs with resolution of clinical signs seen in 17 dogs (7). Also, stereotactic radiotherapy led to significant tumour volume reduction which may improve clinical signs in symptomatic dogs (8).

In our case the chemodectoma caused a life-threatening situation due to marked circumferential narrowing of the lower trachea leading to severe hypercapnia. Hypercapnia is a commonly seen complication during anaesthesia in companion animals (1); however a small increase [<55 mmHg (7.3 kPa)] above the normal ETCO_2 tension of 35 - 45 mmHg (4.7 - 6.0 kPa) is often tolerated (2). In this case the hypercapnia was severe from the start of GA at 99 mmHg (13.2 kPa) and increased gradually as the GA progressed. Manual IPPV was ineffective, although a ventilator might have successfully reduced the ETCO_2 tension because higher airway pressure could have been generated. Hypercapnia has multiple physiological effects, including respiratory acidosis, systemic vasodilatation, pulmonary vasoconstriction, increased cerebral blood flow, sedation leading to narcosis at high values [90 - 120 mmHg (12.0 - 16.0 kPa)], increased circulating adrenaline and noradrenaline concentrations and direct depressant effects on cardiac myocytes (9). In this case the high ETCO_2 tension was related to severe hypoventilation caused by the tracheal narrowing. Causes of hypercapnia other than hypoventilation include i) increased fraction of inspired CO_2 , ii) increased production of CO_2 and finally iii) increased physiological dead space (3). The hypercapnia worsened during anaesthesia, which was probably due to depression of the ventilatory response to hypercapnia caused by anaesthetic drugs (9). The cat could also have been chronically hypercapnic, the central chemoreceptors can become desensitised to hypercapnia when the pH of the cerebrospinal fluid returns to normal due to bicarbonate shift (9), which combined with an inspired oxygen fraction of 94% could have reduced

ventilatory drive further.

Anaesthetic equipment faults can also lead to hypercapnia. Hypercapnia has been reported in a cat due to a defect in the inner tube of a Bain breathing system (10); and in 2 dogs and a cat due to malfunction of the unidirectional valves in a circle breathing system (11). In this case the anaesthetic machine and breathing system had been thoroughly checked and no faults were noted. The fresh gas flow in this case was adequate as confirmed by the lack of rebreathing indicated by capnography. Due to the relatively short duration of GA and movement between different areas of the hospital an arterial sample for arterial blood gas analysis was not obtained before euthanasia. This would have provided useful information including arterial blood pH, arterial partial pressure of CO₂ (PaCO₂) and arterial partial pressure of oxygen (PaO₂) (12). Furthermore, evaluation of the pH and bicarbonate could have given information on the chronicity of the hypercapnia.

Tracheal stenosis is rare in cats and airway management can be challenging and potentially lifesaving. Narrowing of the trachea reduces space within the airway and leads to increased resistance and reduced airflow (13). Other potential causes include: i) stenosis after traumatic intubation (14), ii) stenosis after tracheotomy (14, 15) and iii) tracheal neoplasia (14, 16, 17). Successful management of tracheal resection due to neoplasia (18) and tracheal rupture (19, 20) in cats has been reported. These cases were managed with different techniques including a 3 phased approach to ventilation adapted to 3 different stages of surgical intervention, enabling maintenance of anaesthesia with inhalation agents (18), one lung ventilation with total IV anaesthesia (19) and jet ventilation via a feeding tube (20). One study in humans describes different airway management techniques for placement of a tracheal stent, namely cuffed ETT, laryngeal mask airway, jet ventilation and veno-arterial extracorporeal membrane oxygenation (21). Airway management in these cases will also depend on the site of the obstruction e.g whether the lesion is situated within the intra-thoracic or extra-thoracic portion of the trachea.

One challenge in this case, other than those intrinsic to the presentation itself (i.e. suspected tracheal foreign body that was actually extramural compression by a chemodectoma, difficult

airway management and severe hypercapnia), was that the patient presented out of hours. It is well known that anaesthetic mortality in cats has an increased odds ratio of 1.6 for both scheduled to urgent cases and urgent to emergency cases (22). This is due to the animal's health status (ASA IV-E in this case) and other factors including lack of personnel and tiredness. In retrospect, having a ventilator could have allowed effective continuous IPPV to treat the hypercapnia. A change in case management was necessary because the cat acutely deteriorated. Anaesthetists must be prepared to alter plans at short notice when presented with a rapidly changing clinical picture. This is especially important in emergency situations.

In summary this is the first report of tracheal narrowing caused by a chemodectoma leading to severe hypercapnia in a cat. Furthermore, it highlights that airway management and ventilation can be challenging in these cases. These are important considerations, especially in cases that present as out of hours emergencies where acute deterioration and unexpected primary disease processes can make planning difficult.

LEARNING POINTS/TAKE HOME MESSAGES 3 to 5 bullet points – this is a required field

- Tumours causing mechanical compression of the trachea may cause airway compromise.
- Although chemodectomas are rare in cats they are a potential cause of respiratory distress through a variety of mechanisms.
- Severe hypercapnia is a potential complication in these cases and effective IPPV may be required to prevent dangerous elevations in ETCO₂ tension.

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FIGURE/VIDEO CAPTIONS *figures should NOT be embedded in this document*

FIGURE 1

Soft tissue reconstruction CT images after contrast medium administration in transverse (A), sagittal (B) and dorsal (C) planes. The transverse plane shown is at the level of the fifth thoracic vertebra, where the maximal tracheal narrowing was present. The trachea is shown by the red arrow and the mass is outlined by the yellow arrows. Note air-distended oesophagus (asterisk).

OWNER'S PERSPECTIVE *Optional*

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