Anaesthesia for feline tracheal avulsion surgery – the nurse's role: a case discussion

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ABSTRACT: Tracheal avulsion (TA) is the complete severance of the intrathoracic trachea and in cats is generally attributed to blunt-force trauma resulting in hyperflexion of the neck, often following a road traffic accident (RTA). Some cats possibly suffer a tracheal avulsion which is never diagnosed as they die at the roadside from concurrent fatal injuries. This article will explore the signs, diagnosis and surgical treatment of TA, and specifically the nurse's role during anaesthesia. The discussion involves the case of a 2-year-old male neutered domestic short hair who presented to the clinic with dyspnoea 2 weeks after an RTA.

Keywords: trachea; avulsion; IPPV

Introduction

In the referral setting, veterinary nurses may be tasked with monitoring the anaesthesia for complex and critical cases, some of which may not have been encountered by the veterinary team before. The patient described in this report is Boo, a male neutered domestic short-haired cat weighing 4.5 kg. He presented 2 weeks following initial treatment for a road traffic accident (RTA); a tracheal avulsion (TA) was later diagnosed and required surgical repair.

Tracheal avulsion

The trachea extends from the larynx to the carina and enables the movement of air into and out of the lower airway. It comprises of 38–43 C-shaped hyaline cartilage rings (Roach & Krahwinkel, 2009) and in cats is widely known in the veterinary field to be a delicate structure; particular care should be taken when performing endotracheal intubation in this species.

Intrathoracic TA is caused by a sudden blunt force which causes a whiplash extension of the neck resulting in tearing of the trachea cranial to the carina; the cranial aspect of the trachea is fixed and stronger than the more caudal part (Roach & Krahwinkel, 2009). There may be a partial circumferential tear or a complete avulsion and in some cases the tracheal lumen is maintained by the intact peritracheal adventitial tissue or by the thickening of mediastinal tissue, making the injury survivable. However, over time the ends of the avulsed trachea become inflamed and start to granulate, causing stenosis; the animal will become less tolerant to exercise and display typical signs of dyspnoea (Burton & Monnet, 2005; Roach & Krahwinkel, 2009).

Signs and diagnosis

Signs of TA include either acute-onset dyspnoea immediately following the traumatic event or an insidious onset of worsening dyspnoea over the days or weeks after the incident. Diagnosis can be made via a lateral thoracic radiograph on which discontinuity of the tracheal wall can be appreciated (Figure 1). Other thoracic injuries may be evident, i.e. fractured ribs in the acute patient, but more chronically affected animals may not have any persisting problems other than the respiratory signs (Burton & Monnet, 2005).

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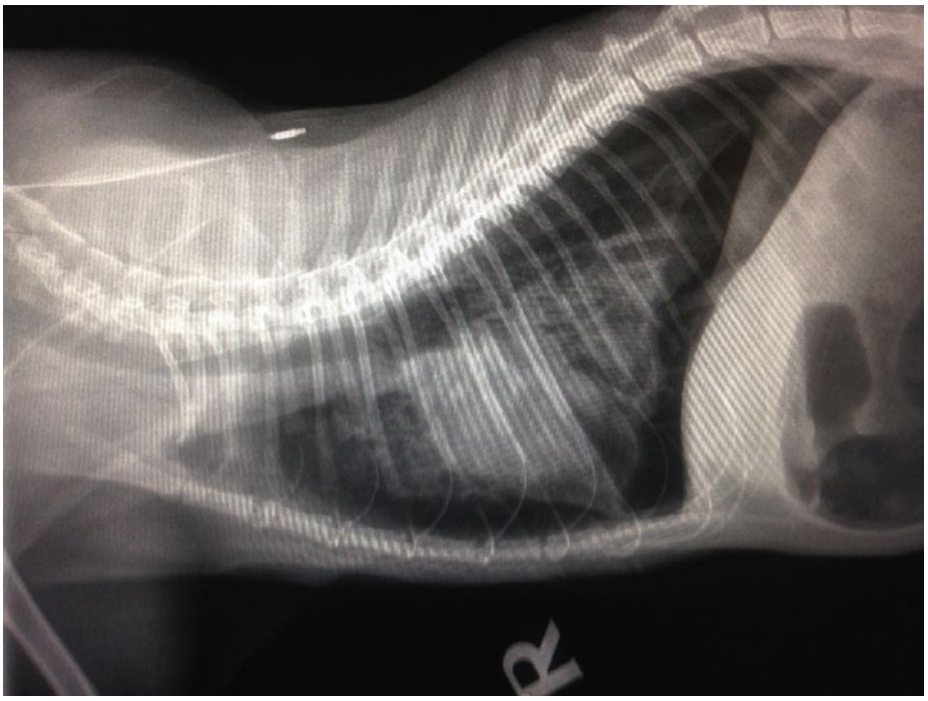

Figure 1. Radiograph showing the tracheal avulsion at the level of the sixth rib.

Treatment

In cases of severe dyspnoea the animal requires rapid induction of anaesthesia and endotracheal intubation – even into the damaged trachea; once stabilised, further investigations and interventions can be carried out (Kirpensteijn, 2006). In cases of TA great care with intermittent positive pressure ventilation (IPPV) is essential to avoid rupturing the 'pseudoairway' that has formed from the tracheal lumen. In cases of TA, surgery is required to remove the damaged segment and perform an anastomosis; an accurate and meticulous surgical technique is vital to encourage an optimal outcome (Fossum, 2013).

Case discussion

Boo was admitted for investigations into dyspnoea as he would begin open-mouth breathing following exertion and at rest would produce respiratory noise. His significant history was an RTA 2 weeks prior to presentation whereby he was hospitalised for 2 days. Boo had scuffed nails, a small amount of haemorrhage from his left nostril, a scrape to his chin and a small scrape lesion on his left hock. He was radiographed under mild sedation and with no bone damage detected he was discharged home with a prescription of oral meloxicam and amoxicillin/clavulanic acid.

Investigations into his dyspnoea some time later revealed the presence of a TA injury with a pseudoairway visible on the radiograph. This was thought to have occurred at the time of the RTA but was not identified on initial survey radiographs. He was scheduled for surgery with the soft-tissue specialist to repair the damage.

Nurse's role

The surgery team had a discussion prior to the procedure to ensure the VS and VNs involved were fully prepared, knew the plan and understood the potential problems that may arise.

The main complications that were discussed included the following.

- Tracheal rupture upon intubation and ventilation; it was agreed gentle manual IPPV would be initiated immediately. A manometer connected to the breathing system allowed the pressure in the lungs to be monitored. Peak airway pressures should not exceed 20 cmH_2O and in healthy animals usually 12–15 cmH_2O is adequate (Hammond & Murison, 2016). It was agreed in this case that the pressure be reduced to 8–10 cmH_2O initially at a rate of 10–12 breaths per minute; this would be guided by capnography to ensure normocapnia, which would confirm an adequate respiration rate and thus effective ventilation.
- Cardiopulmonary arrest (potentially at any given moment). Pre-drawn doses of adrenaline and atropine were to hand and all team members were aware an arrest could happen and briefed on their role.
- Haemorrhage/hypotension during surgery. Crystalloid fluid rates, colloid rates and dopamine doses were pre-calculated and an extra infusion pump and syringe driver were to hand should more than one infusion be required (Figure 2).

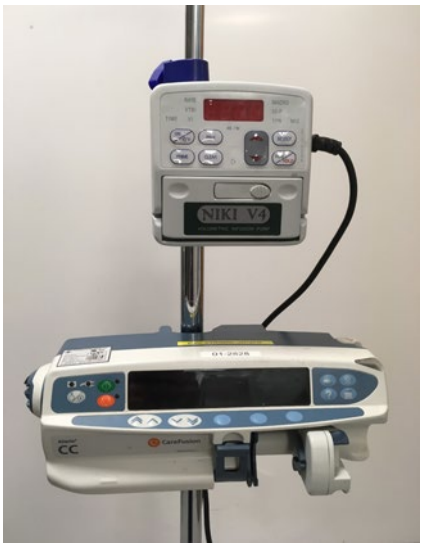

Figure 2. Infusion pump and syringe driver.

The following preparations were undertaken by the VN anaesthetist.

Boo was pre-medicated with an intramuscular (IM) injection of 0.3 mg/kg methadone and 0.01 mg/kg acepromazine. An intravenous (IV) catheter was aseptically placed into the right cephalic vein. Intravenous fluid therapy (IVFT) was Hartmann's crystalloid solution at an initial rate of 3 ml/kg/h. Due to the nature of the surgery which involved a lateral thoracotomy, a 2–10 µg/kg/h fentanyl and 2–10 µg/kg/min ketamine constant rate infusion (CRI) dose chart was calculated and a 2–15 µg/kg/min dopamine CRI dose chart was also prepared. Emergency drugs were calculated and were noted on the anaesthetic monitoring chart and theatre window. A pre-drawn syringe of adrenaline and atropine was prepared, labelled and placed on the anaesthetic trolley. The anaesthetic machine, breathing circuit and mechanical ventilator were all checked for the correct configuration and leaks; the multi-parameter monitor was calibrated and all leads organised neatly for rapid application. A syringe driver and spare fluid pump was placed on the anaesthetic trolley and the emergency drugs box was inspected to ensure it was fully stocked and the drugs were within their expiry date. A Bair Hugger warming device and blanket was situated in the theatre and heat pads with

Figure 3. Penlon Nuffield Series 200 ventilator.

a couple of layers of bubble wrap were ready to help maintain body temperature. A tray for induction was situated in the pre-operative room and included 0.5 mg/kg alfaxalone, a 20 mg/kg dose of amoxicillin/clavulanic acid, intubeaze local anaesthetic spray, a laryngoscope, a 4.0-mm endotracheal tube (ETT) without a cuff and an ETT tie. A second ETT had been sterilised and put in theatre for the surgeon to insert intra-operatively. All surgical site cleaning materials (i.e. a 50:50 dilution of chlorhexidine gluconate and water and sterile gauze swabs) and surgical instruments (i.e. soft tissue pack, thoracic pack, oscillating saw, retractors, diathermy, suction, to name a few) had been prepared.

Boo was induced and an ETT was placed into his trachea and immediately connected to a capnograph. The VN anaesthetist was required to commence IPPV ventilation immediately; great care was imperative as too much pressure may have ruptured the pseudotracheal lining. This was coupled with the need to provide adequate gas to the lungs through the tracheal deficit, making ventilation particularly challenging. Once in theatre manual IPPV continued and all monitoring equipment was attached. The fentanyl CRI was administered and the VS proceeded to enter the right lateral thorax via the fourth intercostal space. The location of the avulsion was not easy to find and Boo suddenly went into cardiorespiratory arrest; this required a rapid tracheostomy just cranial to the azygous and placement of a sterile ETT which was achieved. Mechanical ventilation using a Penlon Nuffield Series 200 ventilator (Figure 3) was initiated at a pressure of 15 cmH_2O and a respiration rate of 10 breaths per minute; Boo's condition quickly stabilised, enabling the remainder of the procedure to be carried out.

The VS found the ruptured trachea and performed an anastomosis having removed 4–5 tracheal rings. The trachea was leak-tested and was air-tight, a chest drain was inserted and the thoracic cavity closed. The drain was placed to allow for the evacuation of any air that may accumulate following an open thorax surgery; removal helps to re-establish the negative pressure within the cavity which is essential for normal ventilation (Aldridge & O'Dwyer, 2013). Boo was moved to the recovery oxygen incubator for observations which included respiration rate and effort, pulse rate and quality, mucous membrane colour and Doppler blood pressure measurements. His temperature was taken but he found this stressful, so it was not repeated too often. A laryngoscope, pre-drawn propofol and emergency drugs, a saline flush and 2.5-mm, 3.0-mm and 3.5-mm ETTs were kept by the incubator in the case of an emergency. A nearby table had been set up with a T-piece attached to the wall-mounted anaesthetic machine to provide 100% oxygen in case of any sudden deterioration. Boo made a steady recovery and the fentanyl and ketamine CRIs were gradually weaned down following regular pain assessments; these were eventually stopped.

Post-operative medication included 0.2–0.3 mg/kg methadone every 4–6 h (following regular pain scoring), 1 ml bupivacaine via the chest drain every 6 h, 0.2 mg/kg meloxicam and antibiotic cover. After the first 12 hs he was given 0.02 mg/kg buprenorphine IV rather than methadone. Boo made a good recovery, his respiration rate and effort remained normal and the thoracic drain was producing no air or fluid. The drain was removed and he was discharged home 48 h after surgery. He had multiple check-up appointments over the next 6 weeks and thankfully made a full recovery.

Conclusion

During a VN's career there may be rare and unusual cases that present requiring procedures that the veterinary team hasn't experienced. A tracheal avulsion is an interesting example of a less-common injury sustained by our small animal patients. In the referral setting there are the equipment, medications, materials and expertise available to undertake these complicated procedures. The VN has a vital role in the process, making a big impact on the outcome for the patient.

This case highlights the need for the VN anaesthetist to discuss the surgical plan with the VS and surgical team to ensure the smooth running of the procedure. It is vital that all involved are aware of the potential complications or 'worst-case scenarios' that may occur and that a plan and preparations are made to deal with a sub-optimal event. Extra training on more advanced aspects of nursing is recommended and involvement in such complicated cases and procedures is highly rewarding, especially when there is a favourable outcome.

Disclosure statement

No potential conflict of interest was reported by the author(s).

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