

LETTER TO THE EDITOR

Cardiopulmonary arrest in a cat as a result of a suspected anaphylactic reaction to an intravenously administered iodinated contrast agent

Since 1950, progress in iodinated contrast media (ICM) has led to the development of new formulations with reduced toxicity. Hunt et al. (2009) reported a prevalence of severe adverse reactions to ICM in humans of 0.153% with a mortality rate of 0.00034%. A study performed in cats showed effects on either heart rate (HR) or systolic arterial pressure (SAP) in 8.77% of cases (Pollard & Puchalski 2008).

A 5-year-old, 3 kg, spayed female Devon rex was referred to our institution for a computed tomography (CT) scan of the head because of a 3-week history of intermittent snoring with an acute onset. No abnormalities were detected on preanaesthetic evaluation and blood work. The patient was classified as ASA (American Society of Anesthesiologists) grade II (a patient with mild systemic disease). A catheter was placed in a cephalic vein. Dexmedetomidine 15 µg kg⁻¹ (Dexdomitor; Elanco, Belgium) and butorphanol 0.2 mg kg⁻¹ (Dolorex; Intervet, Belgium) were administered intravenously (IV). Five minutes later, propofol 6 mg kg⁻¹ (Propofol; Abbott Animal Health, UK) was injected IV over 2 minutes to induce anaesthesia. Lidocaine 0.2 mL (Xylocaine 2%; Astrazeneca, Belgium) was administered topically over the glottis prior to orotracheal intubation with a 3.5-mm internal diameter cuffed endotracheal tube. Isoflurane (Isoflo; Abbott Animal Health, UK) in oxygen was delivered using a rebreathing system. A lactated Ringer's solution (Vetivex VB11A; Dechra, UK) was infused IV at 3 mL kg⁻¹ hour⁻¹. Monitoring during anaesthesia included measurement of heart frequency (HR), determined by direct auscultation (Stethoscope, Littmann 3M) and determination of peripheral

arterial oxygen saturation (SpO₂) and pulse rate (PR) by a portable pulse oximeter (Mindray PM-60 Vet, Germany). The inspired (FIISO) and end-tidal isoflurane percentage (FEISO), end-tidal partial pressure of CO₂ (PECO₂), respiratory rate (f_R) and inspired oxygen percentage (FIO₂) were measured continuously using a multigas analyser (Capnomac Ultima; Datex, Finland). All variables were recorded at 5-minute intervals and remained within respective clinical reference values during the first 30 minutes of anaesthesia. An iodine-containing contrast agent (Ultravist; Bayer, Belgium) was administered IV as a bolus injection (2 mL kg⁻¹). Two minutes after the injection of the contrast, HR and PR dropped to 30 beats minute⁻¹. Atropine 10 µg kg⁻¹ was administered IV. Suddenly, PECO₂ decreased abruptly (to a level of 4 mmHg) and neither HR nor peripheral arterial pulses were detected. The occurrence of an anaphylactic reaction was suspected at this moment. Isoflurane administration was discontinued immediately and external cardiac massage (100 chest compressions minute⁻¹) in combination with a manual intermittent positive pressure ventilation (IPPV) [f_R = 12 breaths minute⁻¹; tidal volume (TV) = 10 mL kg⁻¹; peak inspiratory pressure (PIP) = 12 cm H₂O] with an oxygen flow of 3 L minute⁻¹ was started. Atropine 10 µg kg⁻¹ and epinephrine 0.02 mg kg⁻¹ were administered IV. A palpable femoral pulse and bradycardia were noted and the aforementioned combination of atropine and epinephrine was repeated. At this time, chest compressions were discontinued and PECO₂ values increased slightly but remained below clinically acceptable values (6 mmHg). Auscultation of the thorax revealed severe crackles and serohaemorrhagic fluid was seen inside the endotracheal tube. Manual IPPV was temporarily discontinued to aspirate fluid from the lungs through the endotracheal tube. Atipamezole 75 µg kg⁻¹ (Antisedan; Zoetis, UK), furosemide 2 mg kg⁻¹ (Dimazon; Intervet, UK) and dexamethasone 0.2 mg kg⁻¹ (Rapidexon; Dechra, Belgium) were injected IV. Fifteen minutes after the suspected anaphylactic reaction, 0.2 mg kg⁻¹ promethazine (Phenergan; Sanofi-Aventis, Belgium) was administered. Twenty-five minutes after cardiac arrest, HR and PR were 90 beats minute⁻¹ and PECO₂ rose to 21 mmHg with a

SpO₂ value of 90%. Manual IPPV was discontinued because the patient was able to breathe spontaneously and maintain clinically acceptable values of Pe' CO₂ (≥ 25 mmHg). Fifty minutes later the animal was carried to the intensive care unit, placed in an oxygen cage and monitored carefully. Non life-threatening events occurred during the following post-operative 6 hours, when the patient suffered a second cardiorespiratory arrest with unsuccessful recovery.

Few reports concerning the development of a life-threatening anaphylactic reaction after ICM have been described previously in veterinary patients (Girard & Leece 2010). In our case, a cardiac arrest most likely occurred as a result of an anaphylactic reaction that occurred immediately after the administration of the ICM. Owing to a lack of training of the attending anaesthetist, cardiopulmonary resuscitation was not based entirely on RECOVER guidelines that recommend, for example, different doses of atropine, epinephrine and atipamezole (0.04, 0.01 and 0.1 mg kg⁻¹, respectively) (Fletcher et al. 2012). However, CPR in combination with a symptomatic treatment for anaphylaxis was successfully performed and the patient became stable enough for a transfer to the intensive care unit.

Although the use of steroids before administration of ICM could be considered, in human medicine it is only recommended for patients identified as being at high risk of a reaction to ICM (Bottinor et al. 2013). Furthermore, with the development of newer types of ICM, the incidence of side effects after IV administration of these agents has decreased substantially. Nevertheless, the current report illustrates there is still a potential risk of severe adverse reactions associated with ICM administration. Clinicians play a

critical role in the prevention or reduction of the rate of side effects when working with an ICM. Close haemodynamic monitoring of the patients should be performed to avoid fatal consequences.

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