Received: 1 January 2023 | Revised: 20 April 2023 | Accepted: 6 June 2023

DOI: 10.1002/vrc2.659

Vet Record CaseReports

CASE REPORT

Companion or pet animals

Survival to discharge and outcome of a dog following out-of-hospital cardiac arrest due to asphyxiation

Kathrin Lang | **Abbe H. Crawford** | **Georgina B. F. Hall** | **Karen Humm**

Clinical Science and Services, The Royal Veterinary College, Hatfield, UK

Correspondence
Kathrin Lang, Clinical Science and Services, The Royal Veterinary College, Hatfield, UK.
Email: klang@rvc.ac.uk

Abstract

A 7-month-old dog presented with a 10-minute history of upper respiratory tract obstruction and secondary loss of consciousness in the minute before its arrival to the hospital. The dog was found to be dead on arrival. Cardiopulmonary resuscitation was initiated, which led to successful return of spontaneous circulation 20 minutes later, with subsequent regain of consciousness. Symptomatic therapy and supportive care were provided for 3.5 weeks before discharging the dog into the care of its owners. Despite having made an initial improvement, it was subsequently euthanised 4 weeks later due to persistent severe neurological deficits. Although the neurological improvement was not deemed sufficient for the dog to have a good quality of life, this case report highlights that return of spontaneous circulation after out-of-hospital cardiac arrest and a prolonged period of cardiopulmonary resuscitation is feasible, and neurological improvement thereafter is possible.

KEYWORDS
asphyxiation, cardiopulmonary arrest, resuscitation

BACKGROUND

Out-of-hospital cardiac arrest (OHCA) is defined as a loss of functional cardiac mechanical activity in association with an absence of systemic circulation, occurring outside of a hospital setting.[1] Common causes of OHCA in small animals include trauma, asphyxiation and exacerbation of severe critical illness, such as sepsis.[2] Death often results from asphyxiation due to compromise of the major airways leading to severe hypoxemia and subsequent cardiopulmonary arrest (CPA). Reported survival rates of OHCA range from 7% to 30% in people.[1,3,4] Successful resuscitation is time-dependent and early recognition of OHCA, with contact of emergency medical services (EMS), provision of bystander cardiopulmonary resuscitation (CPR) and defibrillation, all play an important role in improving survival rates in people.[5] So far, only one veterinary study has reported return of spontaneous circulation (ROSC) and survival to discharge in dogs and cats suffering OHCA. However, none of the animals suffering OHCA survived to discharge.[6] This discrepancy in outcome between people and dogs/cats has been linked to the cardiopulmonary arrest cardiac rhythm; while in people, ventricular fibrillation (VF) is the most common initial rhythm of cardiac arrest, non-shockable rhythms such as pulseless electrical activity (PEA) and asystole are more common in dogs.[7] In addition, activation of EMS and provision of bystander CPR are still very limited in veterinary medicine, and speed of arrival to hospital is suspected to be slower, likely contributing to lower survival rates in dogs.

Neurological prognostication after ROSC can be challenging, and is especially unreliable in the first 24 hours. The European Resuscitation Council and European Society of Intensive Care Medicine guidelines published in 2021 recommend delaying neurological prognostication for at least 72 hours after ROSC as well as using a multimodal approach, including a combination of clinical examination, neuroimaging, neurophysiological testing and biomarkers.[8] Repeated neurological evaluation of the patient without sedation is paramount to prevent falsely pessimistic predictions and premature discontinuation of life support for patients who might go on to have a neurologically meaningful recovery.[4,8] This case report documents the successful cardiopulmonary resuscitation of a dog after an OHCA due to upper airway obstruction by a foreign body.

CASE PRESENTATION

A 7-month-old, male, entire cocker spaniel presented to a university teaching hospital, with a 10-minute history of signs of asphyxiation and collapse, after snatching a large piece of cheese from the owner's kitchen. The owners reported that the dog had lost consciousness in the minute before their arrival to

the hospital. On presentation, it was unresponsive, and triage assessment revealed CPA.

TREATMENT

Basic CPR was initiated immediately, with external chest compressions performed on the highest point of the thorax, aiming for 100 compressions per minute. Upon attempting endotracheal intubation, a large piece of cheese was visualised in the pharynx causing airway obstruction. Manual removal of the foreign body was performed, and after successful endotracheal intubation, intermittent positive-pressure ventilation was initiated, with 100% oxygen 2 minutes after presentation. Venous catheterisation of the lateral saphenous vein was accomplished at the same time. Venous blood gas analysis was performed, and it revealed a marked mixed acidosis with a pH of 6.997 (base excess of −10.6 mmol/L [reference range: −2 to +2 mmol/L]; HCO_3^- of 13.1 mmol/L [reference range: 18–24 mmol/L]; partial pressure of CO_2 of 84.5 mmHg [reference range: 37–47 mmHg], hyperkalaemia [5.3 mmol/L; reference range: 3.6–4.6 mmol/L], hypercalcaemia [1.46 mmol/L; reference range: 1.13–1.33 mmol/L] and hyperlactatemia [8 mmol/L; reference range: <2.5 mmol/L]). Arterial blood gas sampling was not attempted at that point. Atropine (0.04 mg/kg intravenously [IV]; atropine sulfate 600 μg/mL, Hameln, Germany), initially chosen as an increase in vagal tone secondary to before death was initially deemed possible, was administered 3 minutes after CPR initiation, followed by adrenaline (0.01 mg/kg IV; dilute adrenaline [epinephrine] injection 1:10,000, Martindale Pharma, UK) 4 minutes later. Venous blood gas analysis at that timepoint showed a marked respiratory acidosis. Conventional monitoring, including electrocardiogram (ECG), end-tidal CO_2 ($ETCO_2$) and pulse oximetry, was instituted, and PEA was determined as the first documented rhythm 3 minutes into CPR. Initial $ETCO_2$ was 26 mmHg, and remained above 17 mmHg throughout the course of the CPR. Nine minutes after initiation of CPR, the ECG showed conversion of the cardiac rhythm to VF, and transthoracic biphasic defibrillation at 50 Joules (3.5 J/kg) was performed 2 minutes later. However, no change of rhythm was noted, and a second defibrillation shock was performed after a 2-minute cycle of cardiac compressions, with an increase in energy to 70 Joules (5 J/kg) without successful conversion. Manual compressions were continued for a further 2 minutes, and a third attempt of defibrillation with 70 Joules was conducted subsequently. No response was noted, and another 2-minute cycle of cardiac compressions was performed. Subsequent ECG analysis showed persistent VF and refractory VF was presumed. At that point, medical defibrillation was attempted, with administration of amiodarone (1.5 mg/kg IV; Nexterone 1.5 mg/mL, Baxter Healthcare, USA), and manual compressions were continued for another 2 minutes until the next ECG analysis. No successful conversion was seen, and a fourth defibrillation attempt was made (70 Joules), followed by ongoing cardiac compressions. This resulted in successful conversion of VF to sinus rhythm and ROSC was achieved 20 minutes after initiation of CPR.

Upon ROSC, the dog started breathing spontaneously. However, manual ventilation was continued as the $ETCO_2$ was 80 mmHg. On suctioning of the endotracheal tube, gross pulmonary haemorrhage was noted, and thoracic point-of-care ultrasound revealed multiple B-lines bilaterally, with a shred sign on the right hemithorax, considered to be consistent with pulmonary contusions. Fifteen minutes following ROSC, venous blood gas analysis was performed showing a marked mixed acidosis, with a pH of 6.787 (base excess of −25.1 mmol/L [reference range: −2 to +2 mmol/L] and partial pressure of CO_2 of 63.7 mmHg [reference range: 37–47 mmHg], hyperglycaemia [21.4 mmol/L; reference range: 4.7–7.3 mmol/L] and hyperlactatemia [15 mmol/L; reference range: <2.5 mmol/L]). Bicarbonate deficit was calculated via standard formula (deficit = [base deficit] × [weight in kg] × 0.3), and 9 mL (one third of calculated dose) of sodium bicarbonate (sodium bicarbonate 8.4%, B. Braun, UK) was diluted in saline (1:2) and was administered over 20 minutes to treat the metabolic component of the acidosis. The manual ventilation rate was increased to manage the respiratory acidosis. Mechanical ventilation was discussed with the owner and the recommendation made to pursue this treatment should the ventilation ability of the dog not improve to an acceptable level within an hour. Hypotension with a systolic blood pressure of 60 mmHg was noted on Doppler sphygmomannometry blood pressure assessment, and so a noradrenaline constant-rate infusion (CRI) (0.2 μg/kg/min IV; noradrenaline 1 mg/mL, Hospira, UK) was started alongside intravenous fluid therapy with Hartmann's solution (2 mL/kg/h).

The dog was transferred to the intensive care unit, and approximately 2 hours after establishment of ROSC, the dog started to ventilate appropriately, determined initially by $ETCO_2$, leading to successful extubation 1 hour later. At that time, the dog was hypothermic, with a core temperature of 35.9°C. Targeted temperature management (TTM) was used, refraining from actively warming the dog, but rather allowing the patient to slowly rewarm, with a core temperature goal of less than 37°C for the next 24 hours. For ongoing oxygen

LEARNING POINTS/TAKE-HOME MESSAGES

- This case report highlights the potential for return of spontaneous circulation and neurological improvement after out-of-hospital cardiac arrest and prolonged cardiopulmonary resuscitation efforts.
- Finding an appropriate sedation protocol for patients following return of spontaneous circulation can be challenging, but current literature suggests that dexmedetomidine might be preferable to benzodiazepines.
- Neurological prognostication in canine survivors of cardiopulmonary arrest is difficult, and data addressing this issue and guiding veterinary and owner decision-making are scarce.
- Limited improvement in neurological deficits is possible despite intensive rehabilitation effort, emphasising the urgent need for improved means of prognostication to avoid prolonging care if outcome is likely to be poor.

support, nasal cannulae were placed bilaterally and a rate of 140 mL/kg/min was chosen initially, aiming for a pulse oximetry reading of 94%–98%, as advocated by the current RECOVER guidelines.[9] The dog regained consciousness at approximately 3 hours after ROSC, with a modified Glasgow coma scale (MGCS) of 14/18. The dog began intermittently vocalising, and a dexmedetomidine CRI at 0.25 μg/kg/h (Dexdomitor 0.5 mg/mL, Vetoquinol, UK) was started, which was increased to 1 μg/kg/h over the following 2 hours. Physiotherapy in the form of passive range of motion and recumbency care (turning, bladder assessment and oral care every 4 hours, eye lubrication every 2 hours) was initiated. As the non-invasively measured blood pressure increased to 150 mmHg, the noradrenaline infusion was stopped at 3 hours after ROSC. Continual vocalising episodes and restlessness were treated with midazolam (CRI at 0.2 mg/kg/h; Hypnovel 10 mg/2 mL, Neon Healthcare, UK) and methadone boluses (0.1 mg/kg IV every 4 hours; Synthadon 10 mg/mL, Animalcare, UK) to increase patient comfort and provide analgesia. Antiemetic therapy with maropitant (1 mg/kg IV every 24 hours; Prevomax 10 mg/mL, Dechra Veterinary Products, UK) was administered to limit the risk of aspiration pneumonia. Venous blood gas analysis was repeated 5.5 hours after ROSC, and it reported a mild respiratory acidosis with a pH of 7.290 (base excess of −1 mmol/L [reference range: −2 to +2 mmol/L]; HCO_3^- of 21.9 mmol/L [reference range: 18–24 mmol/L]; partial pressure of CO_2 of 53.2 mmHg). Glucose (5.6 mmol/L [reference range: 4.7–7.3 mmol/L]) and lactate (1.9 mmol/L [reference range: <2.5 mmol/L]) levels had decreased to within normal limits. Arterial blood gas analysis was performed 8 hours after ROSC. A PaO_2 of 125 mmHg, $PaCO_2$ of 43.1 mmHg and near normalisation of the acid–base status (pH of 7.339 [reference range: 7.350–7.470]; base excess of −2.6 mmol/L [reference range: −2 to +2 mmol/L]; HCO_3^- of 22.2 mmol/L [reference range: 18–24 mmol/L]) was seen. The nasal cannulae oxygen flow rate was slightly decreased to 105 mL/kg/min.

Twenty-four hours after CPR, the dog was recumbent and non-responsive to visual and auditory stimuli. There was an absent menace response bilaterally, an absent direct pupillary light reflex (PLR) in the left eye and a slow direct PLR in the right eye. Occasional paddling of all limbs was observed. A midazolam bolus (0.25 mg/kg IV) was administered, and the midazolam CRI was increased (to 0.3 mg/kg/h). In addition, an initial loading dose of levetiracetam of 60 mg/kg IV followed by 20 mg/kg IV every 8 hours (Keppra 100 mg/mL, UCB Pharma, UK) was administered. A naso-oesophageal feeding tube was placed to facilitate nutritional support. Electroencephalography was performed under sedation (midazolam CRI at 0.3 mg/kg/h, dexmedetomidine CRI at 0.5 μg/kg/h, methadone 0.05 mg/kg IV every 4 hours, levetiracetam 25 mg/kg IV every 8 hours) on Day 2 after resuscitation, and revealed no evidence of epileptiform activity. Discontinuous low-voltage background activity of approximately 15 μV was observed. A thoracic left lateral radiograph was performed for confirmation of accurate feeding tube position, which reported diffuse multifocal pulmonary opacities. Non-cardiogenic pulmonary oedema, pulmonary contusions and pneumonia were listed as possible differential diagnoses. Repeated arterial blood gas analysis on room air showed a near normalisation of PaO_2 (78.8 mmHg) and mild hypocapnia ($PaCO_2$ of 34.4 mmHg), and oxygen support was discontinued at that stage.

Seven days after ROSC, the dog was cardiovascularly stable, had been successfully weaned from oxygen support, the midazolam infusion had been discontinued, repeated neurological examinations documented mild improvements and physiotherapy had been initiated. The dog had regained normal PLRs bilaterally, and was able to raise its head and take a few steps with the thoracic limbs when supported. Voluntary urination and defecation were recorded. However, the menace response remained absent bilaterally, and recurrent episodes of marked apparent distress and dysphoria with vocalisation were observed. Repeated thoracic radiography showed a resolution of the previously noticed pulmonary opacities.

At the beginning of the second week, further dysphoric episodes were treated with multiple boluses of midazolam (0.1–0.3 mg/kg IV). Thereafter, the dog's dysphoric episodes were exacerbated, with an increase in frequency and intensity, including severe paddling of all limbs, unmotivated barking and howling. Electroencephalography was repeated under sedation (midazolam 0.3 mg/kg IV, acepromazine 5 μg/kg IV, levetiracetam 25 mg/kg IV every 8 hours), with recording over 4 hours; no epileptiform activity was observed with continuous background activity of approximately 15 μV. At this stage, it was elected to discontinue all sedative and anticonvulsant drugs that might contribute to the observed dysphoric episodes.

Over the following 7 days, the frequency and intensity of the dysphoric episodes decreased. By the end of the second week, further improvements in neurological status had been observed, with improving tetraparesis and the ability to eat a soft diet with assistance.

OUTCOME AND FOLLOW-UP

The patient was discharged to the owner 25 days after ROSC. At this time, the dog had lost a total of 3.5 kg throughout the hospitalisation period from 14.1 to 10.6 kg at discharge.

On re-evaluation 2.5 weeks after hospital discharge, the dog was able to eat more solid food, and the bodyweight had increased to 11.7 kg. Although a persistent tendency to circle to the left was noted, the tetraparesis had further improved and the dog was able to run on non-slippery surfaces. The owners reported that the dog showed signs of excitement on the owner's return home and was also beginning to alert them when it needed to urinate or defecate. On neurological examination, the dog appeared to be more aware of its surroundings, but remained non-visual, and a resting horizontal nystagmus with the fast phase to the right was noted.

Unfortunately, 2 weeks later, no further neurological improvements were seen, and the owners reported an increase in frequency of howling, ongoing requirement of assistance to eat, reluctance to go for walks, frequent compulsive circling to the left and episodes of defecating in the house. Given this severity of persistent neurological deficits despite a prolonged period of intensive rehabilitation, the dog was euthanased 3 days later at the owners' request. On postmortem examination (PME) of the cerebrum and cerebellum, a diffuse, severe cortical necrosis with multifocal neuronal loss and

perivascular cuffing was noted. In addition, a severe, chronic granulomatous pneumonia with numerous nematodes, morphologically consistent with *Angiostrongylus vasorum*, was present.

DISCUSSION

Successful ROSC after OHCA in veterinary patients is rare, and this is the first report of a dog surviving to discharge after experiencing OHCA. In people, a primary cardiac event is the most likely cause of OHCA, and reported survival rates range from 7% to 30%.[1,5] In contrast, in dogs, CPA as a consequence of progressive systemic illness or trauma is far more common, likely diminishing the chances of survival.[2] Pathophysiology and aetiologies of cardiac arrest in children are distinct from adult cardiac arrests, and do not usually result from a primary cardiac cause.[10] Instead, asphyxia is the most common cause of paediatric OHCA.[11] In paediatric CPA, non-shockable rhythms (asystole/PEA) are far more common than shockable rhythms (pulseless ventricular tachycardia/VF) (82% and 7%, respectively)[12]; whereas in adults, shockable rhythms account for 21%–25% of all sudden cardiac deaths.[3,13] In a cross-sectional study evaluating prognostic indicators for dogs and cats with CPA, 61% of dogs had an initial non-shockable rhythm, while VT/VF was diagnosed in 8%. Therefore, it is possible that pathophysiological processes in CPA in dogs resemble paediatric CPA more than sudden cardiac death in adults. Early respiratory support is an important feature of paediatric CPR and is also considered essential in canine CPA victims.[10,14] Overall survival rates in paediatric OHCA are low and range from 6.7% to 10.2%.[10]

In the current case, once conversion of PEA to VF was noted on ECG, electrical biphasic defibrillation at 3.5 J/kg was initiated immediately and the dose was increased by 50% at the next attempt, following current recommendations.[15] However, successful defibrillation could not be achieved and, in line with the current recommendations for veterinary management of refractory VF, amiodarone was administered, and successful conversion to sinus rhythm was achieved after further defibrillation.[15] In children, administration of either amiodarone or lidocaine is recommended for sustained refractory VF.[10] The same applies for dogs, although amiodarone might be the preferable choice, as it outperformed lidocaine in two studies.[15] Amiodarone has been reported to decrease the defibrillation threshold required to convert dogs experiencing prolonged VF,[16] while the use of lidocaine is associated with an increase in the defibrillation threshold.[17]

The aetiology of a cardiac arrest is of clinical significance. It is well established that asphyxial cardiac arrest results in more severe brain injury than ventricular fibrillation cardiac arrest (VFCA) of the same duration.[18,19] Arterial oxygenation in asphyxiation decreases with increasing hypercapnia before the onset of cardiac arrest, whereas oxygenation is usually normal at the onset of VFCA.[18] In people, complete airway obstruction leads to a fall in PaO_2 to 30 mmHg after approximately 2 minutes when breathing room air. At a PaO_2 of 20 mmHg, apnoea occurs and pulselessness ensues at a PaO_2 of 10 mmHg. Therefore, complete airway obstruction when breathing room air causes clinical death in 5–10 minutes.[20] The asphyxiation episode of this dog lasted approximately 10 minutes before CPA ensued and CPR was started, which is in accordance to the reported timeframe in human medicine. The severe cerebrocortical necrosis and spongiosis noted histopathologically were consistent with the proposed hypoxic-ischaemic injury caused by asphyxiation and CPA.

Pre-existing comorbidity can also impact outcome. Severe, chronic infection with *A. vasorum* was reported on PME in the dog. *A. vasorum* is a nematode parasite that is excreted in the faeces of the definitive host as L1 larvae, which are ingested by slugs and snails, serving as intermediate hosts for a temperature-dependent development to infectious L3. Sources of infection for the dog are intermediate hosts, paratenic hosts or contaminated food. Infestation by L3 larvae is followed by a 30-day migration and a two-stage moult before adults appear in the lungs. As the dog in the present case was hospitalised for 3 weeks, subsequently kept under close observation during rehabilitation by the owners, and ambient temperatures in the region from the day of discharge to the day of euthanasia did not surpass 5°C, infection after asphyxiation seems unlikely but cannot be excluded.[21] While the dog did not show any clinical signs consistent with this disease, it might have exacerbated hypoxic brain injury at the time of cardiac arrest. Pulmonary changes on thoracic point-of-care ultrasound after CPR were noted and presumed to be consistent with pulmonary contusions, but infection with *A. vasorum* is a possible alternative.

TTM is a potential treatment strategy after ROSC, where the patient's body temperature is rapidly reduced with the aim of decreasing neurological injury through the minimisation of the secondary injury phase.[4] Mitochondrial dysfunction, cellular excitotoxicity, oxygen-free radical generation, loss of calcium ion homeostasis and apoptosis are mechanisms implicated in neurological damage in the post-CPR phase.[4] Various studies regarding TTM in humans show conflicting results, and appropriate selection of candidates for TTM appears to be important, given that the initial proposed benefit of TTM could not be reproduced in all patient cohorts and adverse effects of hypothermia can occur.[4,22–24] In paediatric patients who suffered asphyxial OHCA and remained comatose (Glasgow Coma Scale ≤8) after ROSC, 72 hours of TTM was associated with improved 1-month survival rate and 6-month neurological outcome compared with normothermia.[11] The RECOVER guidelines for post-arrest care in dogs are in line with these findings and suggest a beneficial effect of mild therapeutic hypothermia (33°C ± 1°C) for more than 12 hours in dogs that remain comatose after resuscitation.[9] Given that the dog in this case report regained consciousness within hours of ROSC with a MGCS of 14, the decision was made to refrain from inducing hypothermia but rather to allow the patient to rewarm on its own and prevent hyperthermia.

Accurate neurological prognostication in canine survivors after CPR is challenging, and veterinary literature addressing this is sparse. However, early and reliable prognostication of neurological outcome in patients achieving ROSC is important for managing owner expectations and guiding their decision-making process. Care must be taken to avoid premature poor prognostication for patients that could potentially have a favourable outcome. Equally, prolonged rehabilitation efforts should be avoided where neurological deficits are likely to remain severe and result in

compromised quality of life. Absent pupillary, corneal and motor responses 3 days after adult cardiac arrest were predictive of poor neurological outcome in comatose patients.[25] In children with asphyxial OHCA, initial serum lactate level less than or equal to 8.88 mmol/L and GCS score 5–8 before therapeutic hypothermia were significantly associated with a 6-month favourable neurological outcome.[26] To date, no single factor has proven sufficiently accurate in prognostication, and a multimodal approach is likely the most helpful strategy.[4,10] Hyperglycaemia after cardiac arrest has been associated with a worse outcome in people, and animal studies have demonstrated worsening of ischaemic brain injury.[27,28] On venous blood gas analysis, hyperglycaemia, although short lived (<6 hours), was present in this dog.

Delirium, in people, is defined as a syndrome of acute onset disturbance of consciousness, inattention and change in cognition. It is well documented in people after cardiac arrest. Development of delirium is associated with increased morbidity and mortality.[29] Delirium in veterinary medicine is not well characterised. However, considering the definition for people, the dog reported in this study did show marked disorientation, vocalisation and markedly reduced awareness of its surroundings, which could be regarded as delirium. Treatment strategies are scarce, and therefore efforts to prevent or minimise delirium are important. Early initiation of physical therapy decreases post-cardiac arrest delirium and improves functional outcomes at discharge.[29] A light plane of sedation should be targeted when possible, as deep sedation has been linked to an increase in delirium and mortality, and has not been associated with improved neurological outcome.[29] Benzodiazepines have been implicated in development and duration of delirium in critically ill people.[30] In contrast, a meta-analysis suggests that dexmedetomidine reduces the incidence of delirium in intensive care patients.[30] A similar observation was made in the current report. At the time of reintroduction of midazolam in the treatment strategy, episodes of delirium were observed to increase in duration and intensity, and after discontinuation of all sedative drugs, a reduction in delirium was noted within 48 hours.

Survival to discharge after prolonged non-anaesthesia-related cardiopulmonary arrest in dogs is rare, and scientific literature is lacking. The present case of the dog experiencing successful ROSC after OHCA demonstrates the feasibility of cardiac rhythm conversion and resuscitation; however, severe hypoxic brain injury prohibited sufficient recovery during an 8-week follow-up period. Future studies of neurological prognostication in these patients are warranted to help clinicians in guiding owner decision-making and implementing appropriate therapeutic treatment strategies.

AUTHOR CONTRIBUTION STATEMENT

Kathrin Lang: writing and original draft. Abbe H. Crawford, Georgina B. F. Hall and Karen Humm: review and editing.

CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

ETHICS STATEMENT

The authors confirm that the ethical policies of the journal, as noted on the journal's author guidelines page, have been adhered to. No ethical approval was required as this is a case report with no original research data.

FUNDING INFORMATION

The authors received no specific funding for this work.

ORCID

Kathrin Lang https://orcid.org/0000-0001-6182-1687
Abbe H. Crawford https://orcid.org/0000-0003-2176-5873
Georgina B. F. Hall https://orcid.org/0000-0001-6643-7262
Karen Humm https://orcid.org/0000-0002-5770-7950

REFERENCES

1. Myat A, Song KJ, Rea T. Out-of-hospital cardiac arrest: current concepts. Lancet. 2018;391(10124):970–9.
2. Boller M, Fletcher DJ. Update on cardiopulmonary resuscitation in small animals. Vet Clin North Am Small Anim Pract. 2020;50(6):1183–202.
3. Meaney PA, Nadkarni VM, Kern KB, Indik JH, Halperin HR, Berg RA. Rhythms and outcomes of adult in-hospital cardiac arrest. Crit Care Med. 2010;38(1):101–8.
4. Roberts BW, Kavi T, Trzeciak S. Cardiac arrest and cardiopulmonary resuscitation. In: Parrillo JE, Dellinger RP, editors. Critical care medicine, principles of diagnosis and management in the adult. 5th ed. Elsevier; 2019. p. 2–11.
5. Rea T, Kudenchuk PJ, Sayre MR, Doll A, Eisenberg M. Out of hospital cardiac arrest: past, present, and future. Resuscitation. 2021;165:101–9.
6. Hofmeister EH, Brainard BM, Egger CM, Kang S. Prognostic indicators for dogs and cats with cardiopulmonary arrest treated by cardiopulmonary cerebral resuscitation at a university teaching hospital. J Am Vet Med Assoc. 2009;235(1):50–7.
7. Gatson BJ, Swift S, Paranjape V. Successful treatment of prolonged refractory ventricular fibrillation in an anesthetized dog. J Vet Emerg Crit Care. 2022;32(1):129–34. https://doi.org/10.1111/vec.13132
8. Nolan JP, Sandroni C, Böttiger BW, Cariou A, Cronberg T, Friberg H, et al. European Resuscitation Council and European Society of Intensive Care Medicine guidelines 2021: post-resuscitation care. Intensive Care Med. 2021;47:369–421.
9. Smarick SD, Haskins SC, Boller M, Fletcher DJ, RECOVER Post-Cardiac Arrest Care Domain Worksheet Authors. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 6: Post-cardiac arrest care. J Vet Emerg Crit Care. 2012;22(Suppl 1):S85–101
10. Topijan AA, Raymond TT, Atkins D, Chan M, Duff JP, Joyner BL, et al. 2020 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care, part 4: pediatric basic and advanced life support. Circulation. 2020;142:S469–523.
11. Lin JJ, Lin CY, Hsia SH, Wang HS, Chiang MC, Lin KL, et al. 72-h Therapeutic hypothermia improves neurological outcomes in pediatric asphyxial out-of-hospital cardiac arrest—an exploratory investigation. Resuscitation. 2018;133:180–6.
12. Atkins DL, Everson-Stewart S, Sears GK, Daya M, Osmond MH, Warden CR. Epidemiology and outcomes from out-of-hospital cardiac arrest in children: the resuscitation outcomes consortium epistry-cardiac arrest. Circulation. 2009;119(11):1484–91.
13. Daya MR, Schmicker RH, Zive DM. Out-of-hospital cardiac arrest survival improving over time: results from the Resuscitation Outcomes Consortium (ROC). Resuscitation. 2015;91:108–15. https://doi.org/10.1016/j.resuscitation.2015.02.003
14. Hopper K, Epstein SE, Fletcher DJ, Boller M, RECOVER Basic Life Support Domain Worksheet Authors. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 3: Basic life support. J Vet Emerg Crit Care. 2012;22(Suppl 1):S26–43.
15. Rozanski EA, Rush JE, Buckley GJ, Fletcher DJ, Boller M, RECOVER Advanced Life Support Domain Worksheet Authors. RECOVER evidence and knowledge gap analysis on veterinary CPR. Part 4: Advanced life support. J Vet Emerg Crit Care. 2012;22(Suppl 1):S44–64.
16. Wu L, Jin Q, Zhang N. The effects of acute amiodarone on short and long-duration ventricular defibrillation threshold in canines. J Cardiovasc Pharmacol. 2011;58(4):432–8.
17. Dorian P, Fain ES, Davy JM. Lidocaine causes a reversible, concentration-dependent increase in defibrillation energy requirements. J Am Coll Cardiol. 1986;8(2):327–32.
18. Uray T, Lamade A, Elmer J, Drabek T, Stezoski JP, Missé A, et al. Phenotyping cardiac arrest: bench and bedside characterization of brain and heart injury based on etiology. Crit Care Med. 2018;46(6):e508–15.

19. Vaagenes P, Safar P, Moossy J, Rao G, Diven W, Ravi C, et al. Asphyxiation versus ventricular fibrillation cardiac arrest in dogs. Differences in cerebral resuscitation effects—a preliminary study. Resuscitation. 1997;35(1):41–52.
20. Safar P, Paradis AN, Weil MH. Asphyxial cardiac arrest. In: Paradis AN, Halperin HR, Kern KB, Wenzel V, Chamberlain DA. Cardiac arrest. the science and practice of resuscitation medicine. 2nd ed. Cambridge University Press; 2010. p. 969–93.
21. Ferdushy T, Kapel CMO, Webster P, Al-Sabi MNS, Gronvold JR. The effect of temperature and host age on the infectivity and development of *Angiostrongylus vasorum* in the slug *Arion lusitanicus*. Parasitol Res. 2010;107(1):147–51.
22. Johnson NJ, Danielson KR, Counts CR, Ruark K, Scruggs S, Hough CL, et al. Targeted temperature management at 33 versus 36 degrees: a retrospective cohort study. Crit Care Med. 2019;48(3):362–9.
23. Kalra R, Arora G, Patel N, Doshi R, Berra L, Arora P, et al. Targeted temperature management after cardiac arrest: systematic review and meta-analyses. Anesth Analg. 2018;24:100400.
24. Granfeldt A, Holmberg MJ, Nolan JP, Soar J, Andersen LW. Targeted temperature management in adult cardiac arrest: Systematic review and metaanalysis. Resuscitation. 2021;167:160–72.
25. Wijdicks EF, Hijdra A, Young GB, Bassetti CL, Wiebe S, Quality Standards Subcommittee of the American Academy of Neurology. Practice parameter: prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. Neurology. 2006;67(2):203–10.
26. Lin JJ, Lin YJ, Hsia SH, Kuo HC, Wang HS, Hsu MH, et al. Early clinical predictors of neurological outcome in children with asphyxial out-of-hospital cardiac arrest treated with therapeutic hypothermia. Frontiers. 2020;7:534.
27. Müllner M, Sterz F, Binder M, Schreiber W, Deimel A, Laggner AN. Blood glucose concentration after cardiopulmonary resuscitation influences functional neurological recovery in human cardiac arrest survivors. J Cereb Blood Flow Metab. 1997;17(4):430–6.
28. Dalecy L, Lundy E, Barton K, Zelenock G. Dextrose containing intravenous fluid impairs outcome and increases death after 8 minutes of cardiac-arrest and resuscitation in dogs. Surgery. 1986;100(3):505–11.
29. Boncyk CS, Rengel KF, Pandharipande PP, Hughes CG. In the ICU – delirium post cardiac arrest. Curr Opin Crit Care. 2019;25(3):218–25.
30. Ng KT, Shubash CJ, Chong JS. The effect of dexmedetomidine on delirium and agitation in patients in intensive care: systematic review and meta-analysis with trial sequential analysis. Anaesthesia. 2019;74(3):380–92.

How to cite this article: Lang K, Crawford AH, Hall GBF, Humm K. Survival to discharge and outcome of a dog following out-of-hospital cardiac arrest due to asphyxiation. Vet Rec Case Rep. 2023;e659. https://doi.org/10.1002/vrc2.659

MULTIPLE-CHOICE QUESTION

Which of the following is the optimum initial treatment for a dog with acute onset of ventricular fibrillation as an arrest rhythm?

POSSIBLE ANSWERS TO MULTIPLE-CHOICE QUESTION

1. Mechanical defibrillation
2. Electrical defibrillation with a monophasic defibrillator
3. Electrical defibrillation with a biphasic defibrillator
4. Amiodarone
5. Lidocaine

CORRECT ANSWER

c. Electrical defibrillation with a biphasic defibrillator Following the current RECOVER guidelines, electrical defibrillation is the treatment of choice when a ventricular fibrillation has been diagnosed. Prompt electrical defibrillation has been associated with a higher rate of ROSC and survival than no defibrillation; biphasic defibrillation is preferred over monophasic defibrillation. Immediate defibrillation is recommended if the duration of ventricular fibrillation is 4 minutes or less, whereas in arrests lasting longer than 4 minutes, one cycle of cardiopulmonary resuscitation before defibrillation may help replenish energy substrates and increase the likelihood of successful defibrillation.14 The RECOVER Initiative is currently working on an update of the existing guidelines.