

Case Report

Pneumoperitoneum Following Cardiopulmonary Resuscitation in a Dog

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Simple Summary

Pneumoperitoneum is defined as the presence of free gas within the abdominal cavity. In veterinary medicine, this finding is most commonly associated with gastrointestinal perforation, whereas pneumoperitoneum after cardiopulmonary resuscitation has been reported in human medicine but not in veterinary medicine. This report describes pneumoperitoneum identified after successful cardiopulmonary resuscitation in a dog undergoing dental surgery. The dog was managed conservatively with intensive monitoring and supportive care, and the gas accumulations progressively resolved without surgical intervention. The dog recovered completely and remained clinically stable during follow-up. To the author's knowledge, this is the first veterinary report of pneumoperitoneum following cardiopulmonary resuscitation complication in a dog and suggests that conservative management may be considered when there is no clear evidence of gastrointestinal perforation or peritonitis.

Abstract

A 14-year-old intact female mongrel dog presented for periodontal disease underwent anesthesia for dental procedures. During surgery, the dog developed cardiopulmonary arrest (CPA) characterized by a sudden drop in end-tidal carbon dioxide and pulseless electrical activity. Cardiopulmonary resuscitation was initiated and return of spontaneous circulation was achieved after 20 min. Post-resuscitation imaging revealed moderate pneumoperitoneum, hepatic portal venous gas, and gas within the caudal vena cava without evidence of gastrointestinal rupture or peritonitis. Conservative management including intensive monitoring and supportive care resulted in progressive resolution of these gas accumulations and complete clinical recovery. This is the first veterinary report to describe pneumoperitoneum following CPR during dental surgery. The case broadens current understanding of nonsurgical etiologies of pneumoperitoneum in small animals and demonstrates that conservative management may be a viable option in selected post-resuscitation patients.

Keywords: dog; pneumoperitoneum; venous air embolism; cardiopulmonary resuscitation; dental surgery



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1. Introduction

Cardiopulmonary resuscitation (CPR) is a life-saving intervention for cardiac arrest but is also associated with a range of complications resulting from chest compressions and positive pressure ventilation [1,2]. In human medicine, post-CPR complications include thoracic injuries such as rib fractures and pneumothorax, as well as less common abdominal complications [2,3]. However, studies evaluating complications associated with CPR in veterinary medicine remain limited, and the full spectrum of CPR-related injuries in veterinary patients has not been clearly characterized.

Pneumoperitoneum, which is defined as the presence of free gas within the peritoneal cavity, is most commonly associated with gastrointestinal perforation in veterinary medicine. Other reported causes include thoracic trauma, barotrauma from mechanical ventilation, the Macklin effect secondary to respiratory disease, and iatrogenic events such as abdominal trocarisation or endoscopic procedures [4,5]. Although pneumoperitoneum following CPR has been sporadically described in human medicine [6,7], to the authors' knowledge, no veterinary cases have been previously documented.

Venous air embolism (VAE) is defined as the entry of air into the venous circulation, which can compromise pulmonary or cardiovascular function depending on the volume and rate of air entry. In veterinary medicine, VAE has been infrequently reported but is recognized as a potential complication of several procedures, including pneumocystography, cryosurgery, laparoscopic interventions, and inadvertent intravenous air injection [8–12]. Notably, a fatal case of VAE has been described in a cat during dental procedures involving high-speed, air-driven handpieces [13]. This study highlights the need for caution when using pressurized gas systems during dental procedures.

This report describes a unique case of a canine patient that developed both pneumoperitoneum and a suspected VAE following cardiopulmonary arrest (CPA) during dental surgery. The aim of this case report is to document this rare clinical scenario, outline the diagnostic and therapeutic approach, and highlight the potential for clinical recovery with timely and appropriate post-resuscitation care.

2. Case Description

A 14-year-old intact female mongrel dog weighing 3.4 kg was presented to the dentistry department of Kyungpook National University's Veterinary Medical Teaching Hospital with a chief complaint of halitosis, hypersalivation, and anorexia. The dog was intolerant to oral handling, preventing any home dental care, and had not received professional dental scaling in the previous two years. On physical examination, the dog was bright, alert, and responsive, and systemic hypertension was noted, with a Doppler-measured systolic arterial blood pressure (SAP) of 200 mmHg. Cardiac auscultation revealed a grade 3/6 systolic murmur best heard over the left apex. Oral examination revealed significant calculus accumulation and gingival inflammation. Based on the clinical signs and oral examination findings, the patient was diagnosed with periodontal disease. General anesthesia was accordingly planned for full-mouth scaling, dental radiography, and extraction of teeth as indicated.

Preanesthetic bloodwork (complete blood count, serum biochemistry, and venous blood gas analysis) was largely within normal limits. A transient elevation in blood lactate (4.9 mmol/L, reference interval [RI] < 2.5 mmol/L) was observed; this normalized (1.6 mmol/L) following fluid therapy. Thoracic radiographs did not reveal significant pulmonary abnormalities but suggested mild left atrial enlargement. Echocardiographic evaluation confirmed myxomatous mitral valve disease; however, there was no evidence of left atrial or ventricular enlargement. Considering the patient's dehydration and the need for fluid therapy, the procedure was scheduled for the following day. During hospitalization,

the patient's vital parameters remained within normal limits, with a Doppler-measured SAP under calm, resting conditions of 120 mmHg.

The following day, the dog was premedicated with morphine (Morphine hydrochloride, Myungmoon Pharm., Seoul, Republic of Korea) (0.3 mg/kg, intramuscular, IM) and midazolam (Midacum injection, Myungmoon Pharm., Seoul, Republic of Korea) (0.2 mg/kg, intravenous, IV). Anesthesia was induced with propofol (Anepol injection, Hana Pharm., Seoul, Republic of Korea) (5 mg/kg, IV) and maintained with isoflurane. Intraoperative monitoring included electrocardiography (ECG), capnography, pulse oximetry, and noninvasive oscillometric blood pressure measurement. Fluid therapy was administered using a balanced crystalloid solution (Plasma-Lyte) (Plaju OP injection, JW Pharm., Gwacheon, Republic of Korea) at 5 mL/kg/hr. Bilateral infraorbital and mandibular nerve blocks were performed using 0.5% bupivacaine (Pucain 0.5% injection, Reyon Pharm., Seoul, Republic of Korea) (1 mg/kg) to provide regional analgesia.

A comprehensive oral examination performed under anesthesia confirmed the presence of generalized advanced periodontal disease including gingival recession and furcation exposure. Full-mouth scaling was performed using an air-driven dental unit (Anytwo unit, Newmediro, Seoul, Republic of Korea), followed by intraoral dental radiography and extraction of severely affected teeth. Bradycardia (heart rate 80 beats/min) was observed intraoperatively and was treated with glycopyrrolate (Mobinul injection, Myungmoon Pharm.) (5 µg/kg, IV). Intermittent hypotension (mean arterial blood pressure (MAP) < 60 mmHg) was also identified and was initially managed with a fluid bolus (10 mL/kg), which was administered slowly over 30 min in consideration of the patient's pre-existing MMVD. Ephedrine (Ephedrine hydrochloride, Jeil Pharm., Seoul, Republic of Korea) (0.1 mg/kg, IV) was administered as needed for subsequent hypotensive episodes. Approximately 150 min after induction, during extraction of right mandibular fourth premolar and first molar, deep alveolar sockets were exposed at the extraction sites. Shortly after, a sudden and marked decrease in end-tidal carbon dioxide (EtCO₂) was observed during the later stages of the procedure, followed by the loss of palpable femoral pulses. The dog subsequently developed CPA characterized by pulseless electrical activity (PEA), prompting immediate CPR, which was initiated while the dog remained in left lateral recumbency.

Resuscitative efforts were initiated according to the RECOVER guidelines [1] and included administration of epinephrine (Epinephrine, Jeil Pharm.) (0.01 mg/kg, IV), atropine (Atropine sulfate hydrate, Jeil Pharm.) (0.04 mg/kg, IV), flumazenil (Flumazenil, Myungmoon Pharm.) (0.01 mg/kg, IV), and naloxone (Naloxone hydrochloride, Reyon Pharm.) (0.04 mg/kg IV). Basic life support was performed using circumferential chest compressions with a two-thumb technique, delivered in 2 min cycles. After three cycles of basic life support, the arrest rhythm changed to pulseless ventricular tachycardia with a heart rate exceeding 200 beats/min. Defibrillation was performed three times, initially with 2 J/kg, followed by two additional shocks at 4 J/kg. However, the arrhythmia persisted despite defibrillation, prompting administration of lidocaine (Lidocaine hydrochloride hydrate, Jeil Pharm.) (2 mg/kg, IV) for antiarrhythmic control. As the CPA had persisted for more than 10 min, venous blood gas analysis was performed, revealing a pH of 7.00 (RI, 7.31–7.46), venous partial pressure of carbon dioxide (PvCO₂) of 90.7 mmHg (RI, 27–50 mmHg), and bicarbonate (HCO₃[−]) of 22.5 mmol/L (RI, 21–28 mmol/L). The patient's potassium concentration was elevated at 7.15 mmol/L (RI, 3.9–5.1 mmol/L). Therefore, calcium gluconate (Calcium gluconate, JW Pharm.) (50 mg/kg, IV) and sodium bicarbonate (Sodium bicarbonate injection 8.4%, Huons, Seongnam, Republic of Korea) (1 mEq/kg, IV) were administered.

Return of spontaneous circulation (ROSC) was achieved approximately 20 min after the onset of CPA, as evidenced by a sustained $\text{EtCO}_2 > 35$ mmHg and the return of a palpable femoral pulse. However, the dog remained hemodynamically unstable, exhibiting ventricular tachycardia and hypotension, with a Doppler SAP measured at 70 mmHg. To manage this arrhythmia, lidocaine (2 mg/kg, IV) was administered, followed by a continuous rate infusion (CRI) at 25 $\mu\text{g/kg/min}$, which resulted in restoration of sinus rhythm. Despite resolution of the arrhythmia, hypotension persisted; therefore, cardiovascular support including dobutamine (Dobutamine hydrochloride, Myungmoon Pharm.) (5 $\mu\text{g/kg/min}$) and norepinephrine (Norepirin injection, Huons) (0.1 $\mu\text{g/kg/min}$) was initiated. Following vasopressor administration, the dorsal pedal artery pulse quality improved, enabling arterial catheterization for direct blood pressure monitoring.

The dog was transferred to the intensive care unit for close monitoring and supportive care. On initial assessment, the dog was mentally dull and exhibited increased respiratory effort. Crackles were auscultated diffusely over the lung fields. Oxygen supplementation was administered via an oxygen hood, providing an estimated fraction of inspired oxygen (FiO_2) of approximately 40%. The dog remained hypothermic (34 °C). Active warming was not initiated, but passive cooling was maintained by keeping the dog in a cool and well-ventilated environment to support neuroprotection. Arterial blood gas analysis revealed metabolic acidosis with the following values: pH of 7.17 (RI, 7.31–7.46), arterial partial pressure of carbon dioxide (PaCO_2) of 45 mmHg (RI, 35–45 mmHg), arterial partial pressure of oxygen (PaO_2) of 80 mmHg (RI, 80–105 mmHg), HCO_3^- of 16.4 mmol/L (RI, 18–24 mmol/L), and base excess of -11.3 mmol/L (RI, -5 – $+5$ mmol/L). Serum electrolytes had returned to within the reference ranges; however, the blood lactate concentration remained elevated at 5.1 mmol/L. Serum biochemistry revealed elevations in alanine aminotransferase (ALT) at 1000 U/L (RI, 17–78 U/L) and aspartate aminotransferase (AST) at 848 U/L (RI, 17–44 U/L) as well as hypoalbuminemia (albumin, 1.8 g/dL; RI, 2.6–4.0 g/dL). In addition, hyperglycemia was documented with a blood glucose concentration of 362 mg/dL (RI, 60–110 mg/dL). Thoracic and abdominal radiographs obtained post-resuscitation revealed the presence of free abdominal gas, consistent with pneumoperitoneum (Figure 1). To further evaluate possible abdominal causes, non-contrast computed tomography (CT) (Alexion16 TSX-034A, Toshiba, Kawasaki, Japan) was performed. At the time of CT, the dog was obtunded, showed no voluntary movement, and remained clinically unstable while receiving ongoing vasopressor support. Therefore, the CT examination was performed without additional anesthesia. Given the dog's critical status and need for ongoing vasopressor support, no iodinated contrast agent was administered due to concerns regarding potential renal compromise.

The CT scan demonstrated moderate pneumoperitoneum with multifocal gas accumulations distributed throughout the abdominal cavity, particularly in the left dorsocranial abdomen and surrounding the gastric serosa (Figure 2). In addition, hepatic portal venous gas was observed within both the main portal vein and intrahepatic branches as well as in the caudal vena cava (Figure 3). Pulmonary findings included diffuse interstitial and alveolar opacities affecting multiple lung lobes as well as right-sided rib fractures involving the third to fifth ribs.

Following diagnostic evaluation, the patient was hospitalized for ongoing stabilization and intensive care monitoring. Particular focus was placed on the evaluation of pneumoperitoneum, including assessment of the distribution and extent of free gas, and the identification of potential sources such as gastrointestinal perforation. In addition, the patient's body temperature was serially monitored using a rectal thermometer, and the dog was maintained in a hypothermic state for approximately 12 h. Thereafter, the temperature gradually returned to the normal range without active intervention, coinciding

with improved mentation. By the second day of hospitalization, the dog's blood pressure had normalized, allowing gradual tapering and subsequent discontinuation of vasopressor therapy. Serial thoracic and abdominal radiographs revealed a progressive reduction in the volume of free abdominal gas and resolution of the pulmonary infiltrates (Figure 4). The previously abnormal values on blood gas analysis and serum biochemistry including lactate and albumin levels concurrently returned to within reference ranges during the hospitalization period. By the third day of hospitalization, the dog exhibited notable improvement in appetite and activity concentrations. The dog was discharged the following day with stable vital signs and no apparent complications. A follow-up examination conducted three days after discharge revealed no abnormalities. At an eight-month follow-up via telephone, the owner reported the dog remained clinically stable.

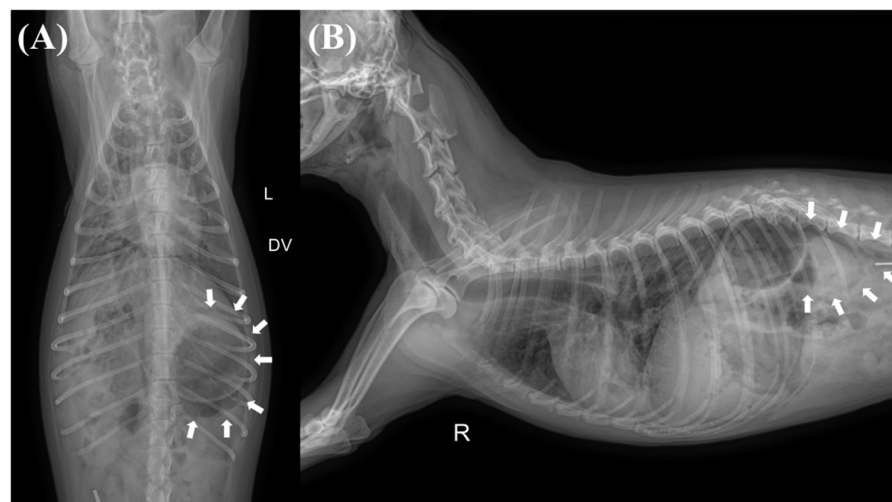


Figure 1. Thoracic radiographs obtained after return of spontaneous circulation (ROSC). (A) Dorsoventral view and (B) right lateral view showing free abdominal gas (white arrows), located around the stomach and within the retroperitoneum. Pulmonary findings included diffuse interstitial and alveolar opacities affecting multiple lung lobes as well as right-sided rib fractures involving the third to fifth ribs.

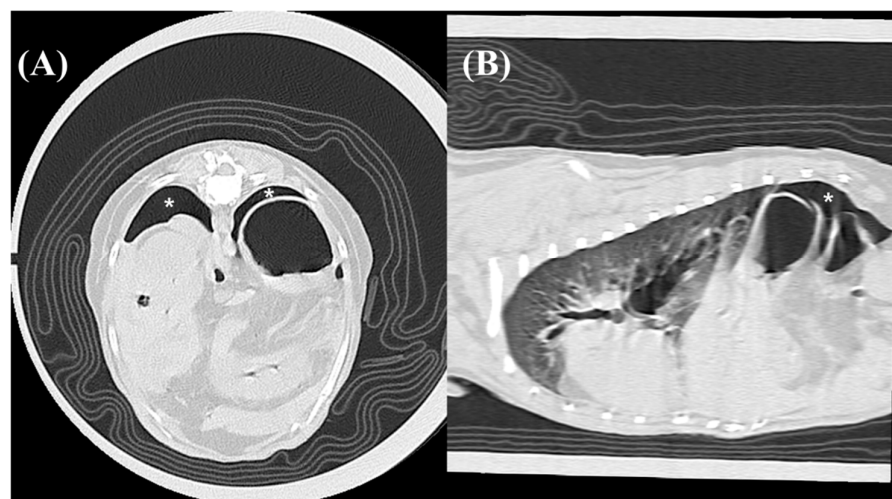


Figure 2. Computed tomography (CT) images in a lung window after return of spontaneous circulation (ROSC). Transverse view (A) and sagittal view (B) showing free abdominal gas (asterisks) consistent with pneumoperitoneum located adjacent to the stomach and within the retroperitoneum.

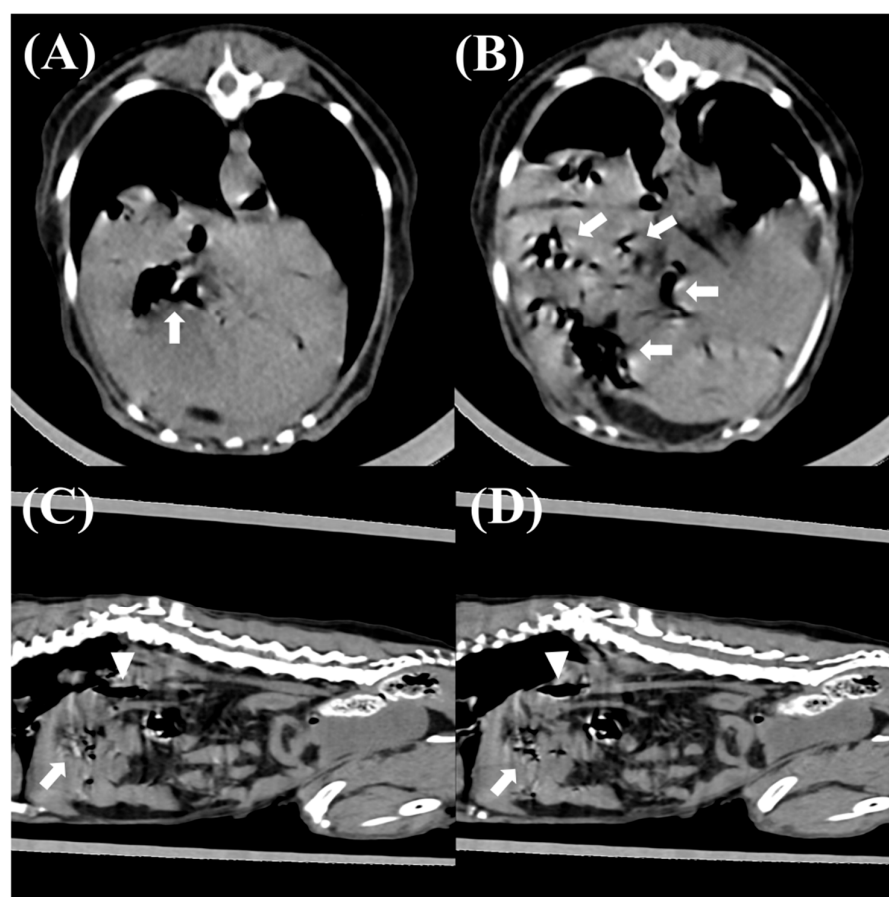


Figure 3. Non-contrast computed tomography (CT) images after return of spontaneous circulation (ROSC). Transverse views (A,B) and sagittal views (C,D) showing gas foci within the hepatic parenchyma (white arrows) and caudal vena cava (white arrow heads).

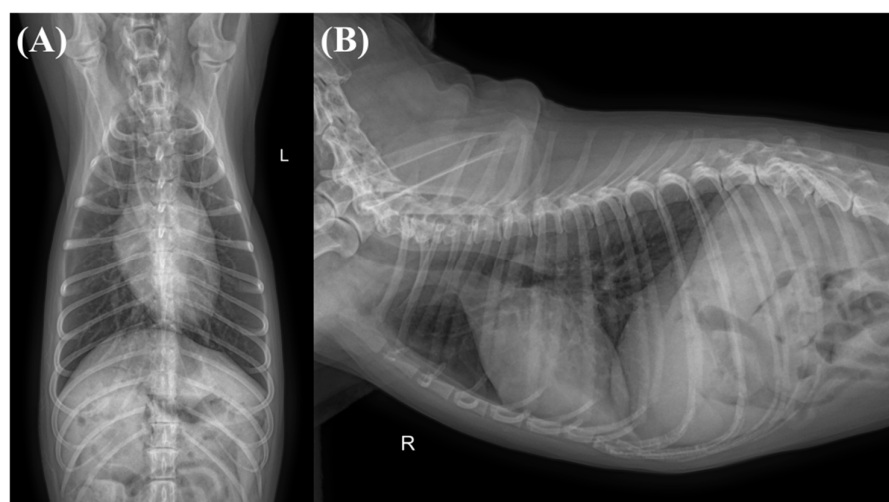


Figure 4. Thoracic radiographs obtained on the third day of hospitalization. Ventrodorsal view (A) and right lateral view (B) showing marked resolution of the previously identified pneumoperitoneum and pulmonary alveolar–interstitial pattern.

3. Discussion

Pneumoperitoneum following CPR is an uncommon but reported phenomenon in human medicine; however, this condition remains poorly characterized in veterinary patients. The proposed pathophysiological mechanisms include alveolar rupture secondary

to excessive intrathoracic pressure from positive-pressure ventilation or vigorous chest compressions, allowing air to dissect along perivascular or fascial planes from the mediastinum into the retroperitoneum and ultimately the peritoneal cavity [7,14]. In addition, gastrointestinal micro-perforation, particularly in ischemic or compromised tissues, has also been suggested as a potential cause of pneumoperitoneum; however, such lesions may be subtle and radiographically occult [15]. In the present case, pneumoperitoneum was incidentally detected on post-resuscitation imaging. No overt evidence of gastrointestinal rupture, such as free peritoneal fluid, bowel wall discontinuity, or clinical signs of peritonitis, was identified on CT or during subsequent clinical observation. Furthermore, there was no radiographic evidence or clinical indication of pneumothorax or pneumomediastinum, which might support a pressure-related origin. Therefore, although CPR-related barotrauma, air dissection, or occult gastrointestinal micro-perforation were considered possible mechanisms, the precise cause and timing of pneumoperitoneum could not be determined. This represents an important limitation of the present report, and the proposed mechanisms should be interpreted cautiously.

Management of pneumoperitoneum requires differentiation between surgical and nonsurgical causes, as gastrointestinal perforation remains the most common and life-threatening etiology in both human and veterinary medicine [4,16]. In both, surgical exploration is recommended when perforation of a hollow viscus is suspected because of the high risk of septic peritonitis and associated mortality [16,17]. In human medicine, nonsurgical pneumoperitoneum has been described in association with positive-pressure ventilation, CPR, and thoracic pathology, and these cases are often managed conservatively when peritoneal signs are absent [6,14,16]. In veterinary medicine, however, such reports are rare; only a single case of thoracic trauma-associated pneumoperitoneum successfully managed without surgery has been reported [18].

In the present case, conservative management rather than exploratory laparotomy was selected because there was no free peritoneal fluid, and CT did not reveal bowel wall discontinuity or other overt evidence of gastrointestinal rupture. The dog also showed no progressive abdominal signs or clinical findings suggestive of peritonitis during hospitalization, and serial radiographs demonstrated progressive resolution of the pneumoperitoneum. These findings supported continued intensive monitoring and supportive care, demonstrating that while surgical intervention remains essential when perforation is suspected, conservative treatment can be appropriate in selected post-resuscitation patients.

Several factors may have contributed to the development of CPA in the present case. Anesthetic factors and the patient's geriatric status may have contributed to decreased cardiovascular reserve and increased susceptibility to hemodynamic instability [19]; however, the abrupt deterioration observed suggests the presence of an additional acute process. In this case, venous air embolism (VAE) was considered a potential contributing factor. A sudden decrease in EtCO₂ was observed immediately prior to CPA, a finding that has been described in previous veterinary reports in association with VAE [12,20]. In addition, gas was identified within the venous system on post-resuscitation diagnostic imaging, suggesting the presence of VAE. Although inadvertent intravascular air introduction or the development of venous gas secondary to CPR cannot be completely excluded, the use of an air-driven dental unit may represent a more likely source, as pressurized air has been reported to enter the venous system through disrupted mucosa or exposed sockets during dental procedures. Similar mechanisms have been proposed in previous case reports describing VAE associated with dental interventions [13]. However, the diagnosis of VAE remains challenging in veterinary patients and is often based on indirect or retrospective findings, which should be considered when interpreting the findings in the present case.

Body positioning during CPR may influence not only the hemodynamic effectiveness of compressions but also the distribution of intravascular gas when present [21,22]. An experimental study in dogs demonstrated that body position significantly affects the cardiopulmonary consequences of VAE, with the left lateral position reducing the migration of air into the pulmonary outflow tract compared with other positions. The authors hypothesized that gravitational effects and anatomical orientation in the left lateral position might impede the coalescence of air in the right heart or pulmonary artery, potentially mitigating hemodynamic compromise [23]. In the present case, CPR was performed while the patient was positioned in left lateral recumbency for a dental procedure, which may have influenced the clinical course. Although the exact impact of positioning cannot be determined, these findings suggest that patient positioning during CPR may have physiological relevance, particularly in cases where intravascular gas is present or suspected. The RECOVER guidelines do not presently specify which lateral recumbency to use [1]; however, right lateral recumbency may be preferred if progression to open-chest CPR is anticipated [24].

In the present case, post-resuscitation care was performed in accordance with established recommendations, emphasizing ventilation, oxygenation, hemodynamic stabilization, and neuroprotection [25]. Continuous arterial pressure monitoring and vasoactive infusions were used to maintain adequate perfusion, while passive hypothermia was managed conservatively to support neuroprotection. Active rewarming was avoided to prevent abrupt increases in metabolic demand. The dog's body temperature gradually normalized as its mentation improved, and no neurological abnormalities were observed during hospitalization or follow-up. Although this is a single case report, the observed response to passive rewarming provides valuable insight for post-resuscitation care in veterinary patients.

4. Conclusions

This case highlights the occurrence of pneumoperitoneum in a geriatric canine patient undergoing dental surgery. The dog experienced intraoperative CPA, and subsequent imaging revealed both portal venous gas and pneumoperitoneum. While the precise etiology remains uncertain, to the authors' knowledge, this represents the first veterinary report to document pneumoperitoneum following successful CPR. The presence of intravascular and intraperitoneal gas underscores the importance of considering gas-related pathologies during the post-resuscitation period, even in the absence of overt gastrointestinal or thoracic injury. Early identification of clinical deterioration, timely intervention, and intensive post-resuscitation monitoring were likely critical to the positive outcome. This case also highlights the need for continued vigilance during procedures involving pressurized gas systems and the need for further investigation into post-resuscitation complications in veterinary medicine.

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Informed Consent Statement: Written informed consent was obtained from the owner of the animal for publication of this case report and accompanying images.

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Abbreviations

The following abbreviations are used in this manuscript:

CPR	Cardiopulmonary resuscitation
CPA	Cardiopulmonary arrest
SAP	Systolic arterial blood pressure
ECG	Electrocardiography
MAP	Mean arterial blood pressure
PvCO ₂	Venous partial pressure of carbon dioxide
HCO ₃ [−]	Bicarbonate
ROSC	Return of spontaneous circulation
CRI	Continuous rate infusion
FiO ₂	Fraction of inspired oxygen
P _a CO ₂	Arterial partial pressure of carbon dioxide
P _a O ₂	Arterial partial pressure of oxygen
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
CT	Computed tomography
VAE	Venous air embolism

References

- Burkitt-Creedon, J.M.; Boller, M.; Fletcher, D.J.; Brainard, B.M.; Buckley, G.J.; Epstein, S.E.; Fausak, E.D.; Hopper, K.; Lane, S.L.; Rozanski, E.A. 2024 RECOVER Guidelines: Updated treatment recommendations for CPR in dogs and cats. *J. Vet. Emerg. Crit. Care* **2024**, *34*, 104–123. [[CrossRef](#)] [[PubMed](#)]
- Wigginton, J.G.; Agarwal, S.; Bartos, J.A.; Coute, R.A.; Drennan, I.R.; Haamid, A.; Kudenchuk, P.J.; Link, M.S.; Panchal, A.R.; Pelter, M.M. Part 9: Adult advanced life support: 2025 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation* **2025**, *152*, S538–S577. [[CrossRef](#)] [[PubMed](#)]
- Van Wijck, S.F.; Prins, J.T.; Verhofstad, M.H.; Wijffels, M.M.; Van Lieshout, E.M. Rib fractures and other injuries after cardiopulmonary resuscitation for non-traumatic cardiac arrest: A systematic review and meta-analysis. *Eur. J. Trauma Emerg. Surg.* **2024**, *50*, 1331–1346. [[CrossRef](#)] [[PubMed](#)]
- Saunders, W.B.; Tobias, K.M. Pneumoperitoneum in dogs and cats: 39 cases (1983–2002). *J. Am. Vet. Med. Assoc.* **2003**, *223*, 462–468. [[CrossRef](#)] [[PubMed](#)]
- Dreese, K.; Magidenko, S.; Glenn, N.; Vilaplana Grosso, F.R.; Wolf, J. Pneumoperitoneum suspected to be secondary to the Macklin effect due to severe upper airway disease and pneumonia in a cat. *Vet. Rec. Case Rep.* **2025**, *13*, e70136. [[CrossRef](#)]
- Hargarten, K.M.; Aprahamian, C.; Mateer, J. Pneumoperitoneum as a complication of cardiopulmonary resuscitation. *Am. J. Emerg. Med.* **1988**, *6*, 358–361. [[CrossRef](#)] [[PubMed](#)]
- Mani, V.R.; Pradhan, L.; Gray, S. Development of pneumoperitoneum after CPR. *Int. J. Surg. Case Rep.* **2015**, *6*, 40–42. [[CrossRef](#)] [[PubMed](#)]
- Ackerman, N.; Wingfield, W.E.; Corley, E. Fatal air embolism associated with pneumourethrography and pneumocystography in a dog. *J. Am. Vet. Med. Assoc.* **1972**, *160*, 1616–1618. [[CrossRef](#)]
- Thayer, G.W.; Carrig, C.B.; Evans, A.T. Fatal venous air embolism associated with pneumocystography in a cat. *J. Am. Vet. Med. Assoc.* **1980**, *176*, 643–645. [[CrossRef](#)]
- Gilroy, B.A.; Anson, L.W. Fatal air embolism during anesthesia for laparoscopy in a dog. *J. Am. Vet. Med. Assoc.* **1987**, *190*, 552–554. [[CrossRef](#)]
- Harvey, H. Fatal air embolization associated with cryosurgery in two dogs. *J. Am. Vet. Med. Assoc.* **1978**, *173*, 175–176. [[CrossRef](#)]

12. Mouser, P.J.; Wilson, J.D. Fatal venous air embolism during anesthesia in an apparently healthy adult Chihuahua. *J. Am. Anim. Hosp. Assoc.* **2015**, *51*, 176–179. [[CrossRef](#)] [[PubMed](#)]
13. Gunew, M.; Marshall, R.; Lui, M.; Astley, C. Fatal venous air embolism in a cat undergoing dental extractions. *J. Small Anim. Pract.* **2008**, *49*, 601–604. [[CrossRef](#)] [[PubMed](#)]
14. Habibullah, N.; Soomar, S.M.; Ali, N. Pneumoperitoneum following cardiopulmonary resuscitation: An unusual case. *Int. J. Surg. Case Rep.* **2022**, *99*, 107649. [[CrossRef](#)] [[PubMed](#)]
15. Spoormans, I.; Van Hoorenbeeck, K.; Balliu, L.; Jorens, P.G. Gastric perforation after cardiopulmonary resuscitation: Review of the literature. *Resuscitation* **2010**, *81*, 272–280. [[CrossRef](#)] [[PubMed](#)]
16. Williams, N.; Watkin, D. Spontaneous pneumoperitoneum and other nonsurgical causes of intraperitoneal free gas. *Postgrad. Med. J.* **1997**, *73*, 531–537. [[CrossRef](#)] [[PubMed](#)]
17. Smelstoy, J.A.; Davis, G.J.; Learn, A.E.; Shofer, F.F.; Brown, D.C. Outcome of and prognostic indicators for dogs and cats with pneumoperitoneum and no history of penetrating trauma: 54 cases (1988–2002). *J. Am. Vet. Med. Assoc.* **2004**, *225*, 251–255. [[CrossRef](#)] [[PubMed](#)]
18. Simmonds, S.L.; Whelan, M.F.; Basseches, J. Nonsurgical pneumoperitoneum in a dog secondary to blunt force trauma to the chest. *J. Vet. Emerg. Crit. Care* **2011**, *21*, 552–557. [[CrossRef](#)] [[PubMed](#)]
19. Hughes, J. Anaesthesia for the geriatric dog and cat. *Ir. Vet. J.* **2008**, *61*, 380. [[CrossRef](#)] [[PubMed](#)]
20. de Celis Perez, C.M.; Santos, L.; Murray, A.G. Successful management of a suspected air embolism during anaesthesia in a dog undergoing thoracic limb amputation. *Vet. Anaesth. Analg.* **2025**, *52*, 656–660. [[CrossRef](#)] [[PubMed](#)]
21. Muth, C.M.; Shank, E.S. Gas embolism. *N. Engl. J. Med.* **2000**, *342*, 476–482. [[CrossRef](#)] [[PubMed](#)]
22. Babbs, C.F.; Voorhees, W.; Fitzgerald, K.; Holmes, H.; Geddes, L. Relationship of blood pressure and flow during CPR to chest compression amplitude: Evidence for an effective compression threshold. *Ann. Emerg. Med.* **1983**, *12*, 527–532. [[CrossRef](#)] [[PubMed](#)]
23. Oppenheimer, M.; Durant, T.; Lynch, P. Body position in relation to venous air embolism and the associated cardiovascular-respiratory changes. *Am. J. Med. Sci.* **1953**, *225*, 362–373. [[CrossRef](#)] [[PubMed](#)]
24. Warang, A.M.; Mann, F.; Middleton, J.R.; Wagner-Mann, C.; Branson, K. Comparison of left fourth and fifth intercostal space thoracotomy for open-chest cardiopulmonary resuscitation in dogs. *J. Vet. Emerg. Crit. Care* **2021**, *31*, 331–339. [[CrossRef](#)] [[PubMed](#)]
25. Johnson, C. Enhancing post-cardiac arrest care for dogs and cats: A veterinary technician’s guide to the RECOVER Initiative. *J. Am. Vet. Med. Assoc.* **2025**, *1*, S85–S91. [[CrossRef](#)] [[PubMed](#)]

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