Part 1

Nursing the canine patient with negative pressure pulmonary oedema

DOI 10.1080/17415349.2021.1931605

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ABSTRACT Non-cardiogenic pulmonary oedema can occur in canine patients as a result of various events. Post-obstructive oedema can occur post choking and results in a rapid accumulation of fluid in the extravascular spaces of the lung. Arterial blood gas analysis and intense patient monitoring are vital to determine the need for further intervention. Treatment can include ventilation, oxygen therapy and medication.

Keywords pulmonary, oedema, canine

Introduction

Pulmonary oedema is the accumulation of fluid in the extravascular spaces of the lung (Bouyssou et al., 2017). It can be categorised into cardiogenic or non-cardiogenic, with the latter being less commonly encountered (Glaus, 2012). This article reports on the multidisciplinary team approach to caring for a puppy diagnosed with negative pressure pulmonary oedema, which developed after choking on a treat. This is a form of non-cardiogenic pulmonary oedema that occurs following obstruction of the upper airway.

The case report describes the pathophysiology of the disease, and discusses the effectiveness of nursing interventions based on current evidence. This includes the team approach to triage, patient monitoring and record keeping, ventilation and oxygenation status, and the medication prescribed to the patient.

Case presentation and investigations

Buddy, a 4-month-old border collie, was presented to the referring veterinary practice 24 hours after a choking event. The patient was eating a treat when he collapsed and began choking. Buddy dislodged the obstruction but deteriorated rapidly.

Prior to arrival at the clinic, the patient underwent conscious thoracic radiographs, along with blood analysis at the referring veterinary practice. There were no significant changes on full biochemistry, and complete blood counts. Orthogonal thoracic radiographs revealed a diffuse bilateral alveolar to interstitial pattern. There was no obvious obstruction to the main airways and no cardiac abnormalities.

Patient assessment

The patient was triaged immediately on arrival by the veterinary nurses, while the clinician obtained a history and gained owner consent. The respiratory, cardiovascular, neurological and renal systems were assessed systematically during the primary survey.

Buddy was tachypnoeic with a respiratory rate between 60 and 80 breaths per minute, with an orthopnoeic posture. This was a concern as, in human medicine, tachypnoea is documented as a discrete and early sign of deterioration (Latimer-Jones, 2020). Additionally, the ribcage of paediatric patients is more compliant, resulting in less competent ventilation and increased respiratory effort (Louro et al., 2019). This could lead to worsened hypoxia and ventilatory fatigue (Louro et al., 2019).

On auscultation of the chest, crackles were identified in all lung fields. These sounds are helpful when considering differentials for diagnosis, as crackles could indicate pulmonary oedema, whereas wheezes are the result of airway narrowing, and absent sounds a result of pleural disease (Latimer-Jones, 2020).

The cardiovascular assessment identified tachycardia, weak and narrow peripheral pulses, cyanotic mucous membranes and an extended capillary refill time (CRT). A prolonged CRT is an indicator of poor peripheral perfusion, which was assumed to be because of peripheral vasoconstriction associated with hypovolaemic shock. Buddy was weak and unwilling to stand but had a normal mentation.

Differential diagnoses

The problems included tachypnoea, cyanotic mucous membranes, and a diffuse alveolar to interstitial pattern on radiographs. There were several differentials, including cardiogenic oedema, non-cardiogenic oedema, aspiration pneumonia or haemorrhage within the alveoli. In addition, systemic disease such as sepsis or acute pancreatitis may result in acute respiratory distress syndrome (ARDS), which can cause a marked non-cardiogenic pulmonary oedema (Glaus, 2012). Furthermore, infections such as leptospirosis are well documented to cause a protein-rich non-cardiogenic pulmonary oedema due to vasculitis (Glaus et al., 2010). Haemorrhage may be due to a multitude of conditions including trauma, coagulopathies, parasites such as *Angiostrongylus* and, potentially, neoplasia such as haemangiosarcoma (Powell, 2002).

Differentiating between these options is often done via a review of the clinical history, vaccination status and patient examination (Sumner & Rozanski, 2013). This patient had no audible murmur or dysrhythmia and no radiographic changes to indicate a cardiac cause (Tong & Gonzalez, 2020). In one paper, post-obstructive pulmonary oedema appeared to show a more asymmetrical unilateral presentation compared to neurogenic pulmonary oedema. However, the paper also states that if the condition is severe, then changes appear to be more diffuse and bilateral (Bouyssou et al., 2017).

In this case, the patient had an acute onset of signs after the owners witnessed an incidence of upper airway obstruction and a period of collapse immediately prior to the onset of clinical signs. In addition, younger animals are at a higher risk of hypoxia due to their smaller functional capacity and thus more at risk of developing pulmonary oedema (Louro et al., 2019). Therefore, a presumptive diagnosis was made of negative pressure pulmonary oedema. In retrospect, an electrocardiogram (ECG) and echocardiography could have been indicated, to rule out any cardiac disease (Agudelo & Schanilec, 2015).

Pathophysiology

Pulmonary oedema typically arises due to an increased intravascular hydrostatic pressure or a disturbed vascular permeability (Glaus, 2012). It can also be caused by an altered oncotic pressure gradient or lymphatic drainage (Glaus, 2012).

There are numerous events that may lead to the movement of fluid into the alveoli, including neurogenic oedema post brain-trauma or seizures, electrocution or re-expansion oedema following rapid removal of a pleural effusion or pneumothorax (Bouyssou et al., 2017). The described patient suffered negative pressure pulmonary oedema, secondary to airway obstruction (Louro et al., 2019). This can be caused by brachycephalic upper airway syndrome, laryngeal paralysis, tracheal collapse, endotracheal tube obstruction and strangulation.

During the obstruction, the patient struggled to breathe, resulting in an increased inspiratory effort. This can lead to mechanical ventilatory stress, which damages the pulmonary epithelium and endothelium (Glaus, 2012). The patient became hypoxic, leading to a sympathetic capillary vasoconstriction (Bouyssou et al., 2017). Additionally, the increased inspiratory effort likely resulted in a large negative transpulmonary and interpleural pressure gradient (Louro et al., 2019).

When negative pressure in the thoracic cavity occurs, it can lead to a decrease in venous blood flow to the heart, allowing blood to pool in the pulmonary vessels (Glaus, 2012). This increases the pulmonary intravascular volume which, when coupled with vasoconstriction, results in a significant increase in hydrostatic pressure (Glaus, 2012).

There are limited reports in veterinary literature describing the treatment of such patients. The largest published research was a series of cases describing the radiographic appearance of 23 cases with pulmonary oedema (Boiyssou et al., 2017). Most recently, Louro et al. (2019) described the diagnosis and management of post-anaesthetic non-cardiogenic pulmonary oedema in a dog.

Arterial blood gas analysis

An arterial catheter was aseptically placed into the left dorsal pedal artery to allow for regular arterial blood gas analysis. A sample was taken on admission and then regularly throughout hospitalisation **(Figure 1)**.

PATIENT'S BLOOD GAS ANALYSIS RESULTS

pH – 7.4 (7.350–7.450)

PCO_2 – 44.4 mmHg (35–38 mmHg)

pO_2 – 56.5 mmHg (85–100 mmHg)

$cHCO_3$ – 27.5 mmol/l (15–23 mmol/l)

BE (ecf) – 2.7 mmol/l (–5–0.0 mmol/l)

cSO_2 – 88.8% (90–100%)

Na^+ – 135 mmol/l (139–150 mmol/l)

K^+ – 3.4 mmol/l (3.4–4.9 mmol/l)

Ca^{++} – 1.19 mmol/l (1.12–1.40 mmol/l)

Cl^- – 100 mmol/l (106–127 mmol/l)

TCO_2 – 27.2 mmol/l (17–25 mmol/l)

Hct – 33% (33–50%)

cHgb – 11.1 g/dl (12–17 g/dl)

BE – 2.3 mmol/l (–5–0.0 mmol/l)

Glu – 7.6 mmol/l (3.3–6.4 mmol/l)

Lactate – 1.33 mmol/l (0.6–2.9 mmol/l)

BUN – 18 mmol/l (2.5–9.6 mmol/l)

Urea – 6.5 mmol/l (3.6–9.3 mmol/l)

Crea – 52 mmol/l (44–115 mmol/l)

BUN/Crea – 31.0 mg/µg (0.2–400 mg/µg)

Urea/Crea – 125.1 mmol/l (0.8–1615.4 mmol/l)

Figure 1. Patient's blood gas analysis results, including (in brackets) normal reference ranges for a canine arterial sample.

Arterial blood gas is considered the gold standard for monitoring oxygenation status (Farrell et al., 2019). The partial arterial pressure of oxygen (PaO_2) and carbon dioxide ($PaCO_2$) can indicate lung function.

The PaO_2 represents the oxygen saturation of haemoglobin in a sigmoid relationship according to

the oxygen dissociation curve (Waddel & King, 2018). Haemoglobin is expected to be fully saturated at a PaO_2 between 60 and 70 mmHg (Waddel & King, 2018). Animals with a normal lung function breathing room air typically have a PaO_2 five times greater than the inspired percentage concentration of oxygen (Angel & Seymour, 2015). Therefore, a patient breathing room air (~21% oxygen) should have a PaO_2 of 100 mmHg, whereas a patient breathing 100% oxygen when anaesthetised and intubated should have a PaO_2 of 500 mmHg (Angel & Seymour, 2015). A PaO_2 of less than 55 mmHg is immediately life threatening (Farrell et al., 2019).

Animals in severe respiratory distress are expected to have an increased respiratory rate and effort with hypoxaemia and hypocapnia with $PaCO_2$ values <35 mmHg. However, in this patient there was a marked hypoxaemia (PaO_2 34.3 mmHg) and a relative hypercapnia ($PaCO_2$ 39.0 mmHg).

$PaCO_2$ is the primary indicator for the ventilatory function of a patient. Elevations in $PaCO_2$ typically develop due to hypoventilation as the patient is breathing inefficiently with a reduced alveolar minute ventilation (respiratory rate (RR) × tidal volume (TV)) (Hopper & Powell, 2013). It is important, however, to consider that in haemodynamically unstable patients, CO_2 will accumulate within the venous system due to low flow states and does not indicate an issue with ventilation (Hopper & Powell, 2013). Hypoventilation may be due to multiple issues, either neurological, metabolic or respiratory in origin, and can indicate respiratory fatigue.

In this patient, it was determined the relatively high $PaCO_2$ was a product of a reduced TV, as it has been shown that those with lung parenchymal disease have reduced compliance, which results in a lower TV (Rozanski, 2015). In addition, due to the poor compliance of the lungs, the work of breathing is greatly elevated and consequently respiratory fatigue may develop, resulting in orthopnoea and appearance of fatigue, like that of the patient (Hopper & Powell, 2013). To prevent this, mechanical ventilation was chosen as an intervention.

Further diagnostics

Diagnosis was based on history and clinical examination. Thoracic radiographs were taken at the referring veterinary practice and were not repeated on arrival. On reflection, thoracic ultrasound could have been used as it allows repeated assessment and is a non-invasive imaging method. Continuous ECG monitoring is necessary to monitor heart rate and the presence of dysrhythmias. However, in this case, the puppy was stressed and unwilling to allow ECG pads or clips to be placed.

Part 1 summary

There are numerous causes of non-cardiogenic pulmonary oedema, therefore it is vital to gain a detailed history to guide differential diagnoses. Having an understanding of the pathophysiology can help to guide diagnostic tests such as radiography and blood gas analysis, which will further allow specific treatments to be instigated. Often these patients present dyspnoeic and, to prevent further deterioration, the team must work efficiently and quickly to triage the patient. A whole-team approach proved useful in Buddy's case, where the veterinary nurses and interns assessed the patient while the veterinary clinician gained history and consent. The case study in Part 2 discusses the management of Buddy's airway, including ventilation, oxygen therapy and the medications prescribed.

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