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Pulmonary oedema in a hunting dog: a case report

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ABSTRACT: This case report describes an episode of acute dyspnoea after hunting in a Dachshund dog. Thoracic radiographs confirmed pulmonary oedema. Echocardiography, electrocardiography and cardiac markers were within normal limits. Based on the history, clinical signs and other diagnostic tests this case could have been neurogenic non-cardiogenic pulmonary oedema-like syndrome as described in Swedish dogs also after hunting and probably the first case detected in Central Europe. The dog survived with intense oxygen, diuretic and bronchodilator therapy. The pathological mechanisms of neurogenic non-cardiogenic pulmonary oedema are also discussed.

Keywords: neurogenic lung oedema; diuretic therapy; oxygen; catecholamines

List of abbreviations

ARDS = acute respiratory distress syndrome, **CBC** = complete blood count, **ECG** = electrocardiogram, **NCPO** = non-cardiogenic pulmonary oedema, **NP** = natriuretic peptides, **NT-proBNP** = N-terminal pro-brain natriuretic peptide, **SAS** = sympatico-adrenal system, **VHS** = vertebral heart size

Pulmonary oedema is defined as an increased amount of fluid in the lungs due to increased vascular permeability or elevated hydrostatic pressure. Pulmonary oedema may be cardiogenic or non-cardiogenic in origin. Cardiogenic oedema originates from an increased hydrostatic pressure in the pulmonary capillaries mostly due to left-sided congestive heart failure (Kakouros and Kakouros 2003; Glaus et al. 2010). Non-cardiogenic pulmonary oedema (NCPO) can develop from decreased alveolar pressure, increased vascular permeability or neurogenic disease (Glaus et al. 2010).

NCPO due to decreased alveolar pressure can result from transient obstruction of the upper airways (i.e. laryngeal paralysis, strangulation or foreign bodies). There is probably a combination of negative intra-thoracic pressure, hypoxia, and increased activity of the sympatico-adrenal system that are generally associated with higher vascular resistance and higher pulmonary venous return to the right heart, which increases transmural pressure across the membrane of the alveolar capillaries. Similar mechanisms can explain re-expansion oedema, which can be a complication of pneumothorax or pleural effusion after tapping (Glaus et al. 2010; Bachman and Waldrop 2012). NCPO caused by increased permeability of blood vessels may be associated with acute respiratory distress syndrome (ARDS), which is primarily caused by pathological processes resulting in systemic inflammatory response syndrome leading to an imbalance between pro and anti-inflammatory mediators (Bachman and Waldrop 2012). A large number of pro-inflammatory factors and cells can damage endothelia and induce increased permeability to the alveoli (Kakouros and Kakouros 2003; Bachman and Waldrop 2012). Other factors that can induce this type of NCPO are inhalation of smoke or other toxins, oxygen toxicity, post-transfusion reactions, near drowning and aspiration pneumonia (Glaus et al. 2010). Pulmonary oedema of neurogenic origin may originate from seizures, traumatic brain injuries or electrocution (Drobatz et al. 1996; Kakouros and Kakouros 2003). Massive central sympathetic

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neural stimulation releases large amounts of catecholamines into the bloodstream. The consequences of the release of catecholamines are not entirely clear, but they seem to involve significant pulmonary and peripheral vasoconstriction, which ends in pulmonary and systemic hypertension (Malik 1985; Glaus et al. 2010; Davison et al. 2012). This paper describes a hunting dog that developed pulmonary oedema after chasing prey.

Case description

A two-year-old Dachshund female intact dog was admitted to the Small Animal Clinic at the University of Veterinary and Pharmaceutical Sciences, Brno, Czech Republic with a one day history of acute respiratory distress after hunting. On the preceding day, the dog had undergone 3 h of hunting training, during which time it escaped, and after its return it suffered from dyspnoea lasting the entire night. The owner stated that the dog very probably suffered an attack of prey.

The first evaluation revealed a depressed animal with mildly cyanotic mucous membranes. Tachypnoea (80 rpm), mixed dyspnoea and orthopnoea were observed. Auscultation revealed bilaterally increased broncho-vesicular sounds with the presence of crackles. Careful examination ruled out signs of head or external body trauma and upper airway obstruction. Oximetry showed an oxygen saturation of 78%. Initial management included an indwelling catheter, oxygen therapy, butorphanol (0.2 mg/kg *i.m.*) and etamsylate (10 mg/kg *i.v.*). After a brief stabilisation and slight clinical improvement radiographs of thorax and abdomen were obtained.

Thoracic radiography (Figure 1) revealed a normal heart size (vertebral heart size – VHS 10.5), symmetrical interstitial-to-alveolar lung pattern in the caudal lung lobes and enlarged pulmonary arteries. There was also gastric distention probably due to aerophagia as a result of the laboured respiration. Radiographic differential diagnoses included pulmonary oedema, lung contusion, haemorrhage, pneumonia or diffuse neoplasia. Complete blood count (CBC), biochemistry and coagulation profile showed hyperfibrinogenaemia (5.66 g/l; reference value 1.5–4), shortened thrombin time (7 s; reference value 10–19) and increased ALT activity (1.35 μkat/l; reference value 0.1–1). Fibrinogen, as an acute phase protein, can be increased in a variety of stressful and inflammatory conditions, and this is closely related with the shortened thrombin time. In some cases this condition could cause hypercoagulability and thrombi formation. The patient did not experience haemorrhagic episodes before or during hospitalisation. Based on the fact that the dog performed heavy hunting training and on the radiographic findings, a tentative diagnosis of NCPO was made. Oxygen therapy and etamsylate (TID) were continued and aminophylline (5 mg/kg *i.v.*, TID) and furosemide (4 mg/kg to effect) were added to the therapy.

On the 2nd day the patient's clinical status improved mildly but mild dyspnoea and tachypnoea (60 rpm) with mild signs of orthopnoea was still present and worsened with patient manipulation. Increased broncho-vesicular sounds remained on

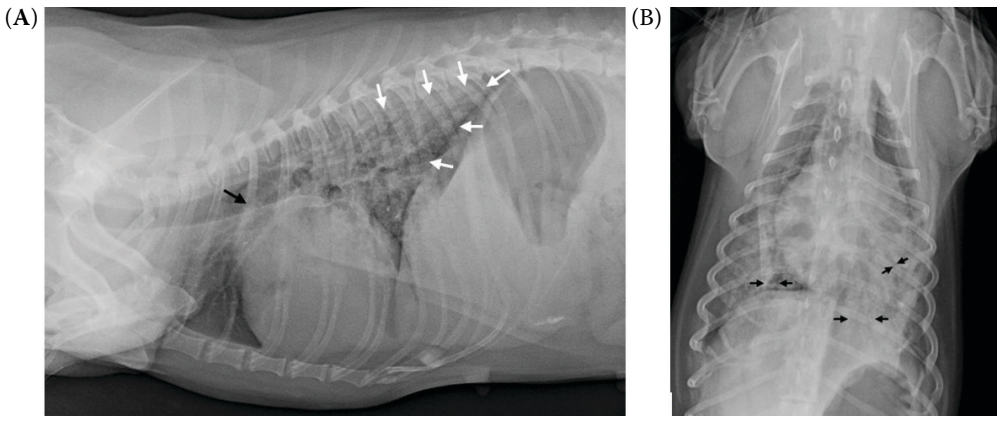

Figure 1. Right lateral and dorso-ventral thoracic radiographs of a two year old female dachshund with respiratory distress. **A**. The right lateral view shows prevailing alveolar pattern in the right caudal lung lobe (white arrows). There is no cardiomegaly (VHS of approximately 10.5). The right cranial lobar pulmonary artery is dilated (black arrow). **B**. Dorso-ventral view shows symmetrical alveolar pulmonary pattern in the right middle and caudal lung lobes and in the left craniocaudal and caudal lung lobes. Also evident are air bronchograms in both caudal lung lobes (bronchus visible between black arrows)

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the auscultation. Oximetry showed an oxygen saturation of 99%. Another CBC showed lymphopenia (768 × 10^9/l; reference value 1000–3600) and monocytosis (1440 × 10^9/l; reference value 0–500) probably as a result of catecholamine release due to stress or neurogenic-mediated mechanisms. Biochemistry indicated mildly increased ALT (1.57 μkat/l) and hypokalaemia (3.5 mmol/l; reference value 3.6–5.5). The increased ALT and hypokalaemia were probably due to the hypovolaemia and diuretic therapy. Electrocardiography (ECG), echocardiography and N-terminal pro-brain natriuretic peptide (NT-proBNP) (158 pmol/l; reference value < 900) were performed with normal findings. Therapy continued unchanged. On the 3rd day of hospitalisation the clinical status noticeably improved. A further NT-pro BNP test was normal (14 pmol/l). Aminophylline and etamsylate were discontinued and furosemide was reduced to 2 mg/kg *i.v.* BID. On the 4th day the dog was discharged with furosemide at 1 mg/kg PO BID for one more week. Follow-ups could not be performed on this dog.

DISCUSSION AND CONCLUSIONS

This case report describes a hunting dog which developed pulmonary oedema due to unclear circumstances. A syndrome of acute dyspnoea occurring during or after hunting has been described in dogs in Sweden (Lord et al. 1975) and it is believed that it could be connected with the neurogenic form of NCPO (Egenvall et al. 2003). Trigger factors in the observed hunting dogs seemed to be transient hypoglycaemia during exercise, stress, excitement and the strenuous exercise of te hunting (Lord et al. 1975; Egenvall et al. 2004). With the exception of hypoglycaemia all these findings were evident in this case. It is suspected that the mechanism of NCPO involves massive central neural sympathetic stimulation in the medulla oblongata (Davison et al. 2012), which promotes the release of a large amount of catecholamines (Malik 1985; Glaus et al. 2010).

Catecholamines can cause significant pulmonary and peripheral vascular changes by provoking a rapid shift of blood to the central circulation mediated through systemic (Lord et al. 1975; Davison et al. 2012; Drobatz and MacIntire 2012) and pulmonary vasoconstriction ("blast theory") (Bachman and Waldrop 2012; Davison et al. 2012), in turn leading to systemic and pulmonary hypertension (Bachman and Waldrop 2012). This increases the pressure in the left atrium as a result of a reduced cardiac output and increased peripheral vascular resistance (Davison et al. 2012), producing an increase in hydrostatic pulmonary pressure and damage to the alveolo-capillar epithelium, which ultimately results in vascular disruption and fluid escape (Bachman and Waldrop 2012). All these mechanisms can be followed by lymphatic vasoconstriction and insufficient lymphatic circulation (Glaus et al. 2010) worsening oedema formation.

Catecholamines can also induce a myocardial "spasm" of the left ventricle causing ischaemia that mainly affects diastolic function creating congestion and oedema (Lord et al. 1975). Furthermore, there is evidence that certain hormones like neuropeptide Y and endothelin-1 can exacerbate oedema formation by increasing pulmonary vascular permeability and vascular pressure (Bachman and Waldrop 2012).

One large study showed that 27% of dogs suffering from NCPO were Dachshunds (Egenvall et al. 2003), which indicates a possible genetic predisposition in this breed. Interestingly, in the medical literature all cases of NCPO have been reported in hunting dogs in the north of Europe. To the authors' knowledge this could be the first description of this pathology in Central Europe (Czech Republic), in a hunting dog. In fact, experienced local hunters have speculated that this respiratory syndrome could be more usual than is reported. A fatal outcome has been noticed in some cases with signs of exercise intolerance and dyspnoea. However, cases of NCPO are poorly documented and very few seek veterinary care (Lord et al. 1975; Egenvall et al. 2003).

Clinical examination of patients with NCPO usually reveals tachypnoea and dyspnoea (Lord et al. 1975; Drobatz et al. 1996; Drobatz and MacIntire 2012). Unfortunately, because the dog escaped and returned already in respiratory distress, other causes like transient obstruction of the upper airways or direct head trauma could have happened, but they were ruled out during the initial clinical examination and hospitalisation. Moreover, there was no history of previous neurological disease in the dog.

Thoracic radiographs are the fastest and most widely available diagnostic method for establishing a diagnosis of pulmonary oedema. NCPO typically exhibits a caudodorsal interstitial-to-alveolar lung pattern, usually with bilateral distribution (Glaus et al. 2010; Bachman and Waldrop 2012; Drobatz and MacIntire 2012). Most of these cases do not exhibit pulmonary

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venous congestion. Reported cases of NCPO in hunting dogs can reveal the presence of transient mild enlargement of the heart according to the VHS, due to the possible catecholamine-mediated shift of blood to the central circulation (Egenvall et al. 2003), and should not be considered reliable evidence of heart disease. Other possible causes of acute dyspnoea with pulmonary infiltrates are lung trauma, ARDS, pneumonia, and pulmonary oedema of cardiogenic origin; most of these potential causes were ruled out in this case by history, blood work and cardiologic examination, with the exception of lung trauma. Echocardiography can also show slight left atrial enlargement explained by increased blood return as a consequence of the central redistribution of blood and depressed left ventricular function (Egenvall et al. 2003). ECG and echocardiographic examination should be performed in all patients to rule out any cardiac disease leading to lung oedema. NCPO can also be diagnosed using computed tomography in humans. This method gives superior accuracy and thus influences the therapeutic approach. Cardiac markers can be used to differentiate cardiogenic pulmonary oedema from NCPO. Natriuretic peptides (NP) are elevated in conditions that produce long-term stretching of the heart muscle like in heart disease (Bachman and Waldrop 2012). In our case, the values of NT-proBNP were within normal ranges during all stages of hospitalisation supporting a non-cardiogenic aetiology.

Therapy of NCPO can involve cage rest, oxygen and medical treatment. The early stages of NCPO exhibit a bronchial pulmonary pattern in thoracic radiographs; according to the recommendations in human medicine the suggested therapy at this stage would be cage (bed) rest. Once the infiltrates advance to the interstitium, patients should be treated additionally with oxygen. Most veterinary patients are admitted with advanced pulmonary infiltrates and pharmacological intervention may be indicated. In addition to the use of cage rest and oxygen, we have observed that some patients benefit from sedation, diuretics, bronchodilation, and in extraordinary situations with positive ventilation. Ventilation in animals is a consideration, but obviously the labour is intensive, expensive and not available in all practices. We regularly administer to those patients other alternatives like diuretics and bronchodilators. Furosemide has been recommended and used as the treatment of choice for both cardiogenic and NCPO in veterinary medicine (Drobatz and MacIntire 2012); though its use remains controversial (Bachman and Waldrop 2012). Bronchodilators could be of benefit to relieve signs of bronchospasm. Other pharmacological interventions, specifically anti-α-adrenergic agents (chlorpromazine), can potentially interrupt the vicious cycle of haemodynamic instability and subsequent respiratory failure. However, confirming the suitability of such agents clearly requires further study (Davison et al. 2012).

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