

## ANGIOSTRONGYLOSIS-RELATED RESTRICTIVE PNEUMOPATHY ASSESSED BY ARTERIAL BLOOD GAS ANALYSIS IN A DOG

Judit CSÖNDES<sup>1\*</sup>, Gábor MAJOROS<sup>2</sup>, Zoltán LAJOS<sup>3</sup>, Roland PSÁDER<sup>4</sup>, Péter VAJDOVICH<sup>1</sup>,  
Ferenc MANCZUR<sup>4</sup> and Ákos MÁTHÉ<sup>4</sup>

<sup>1</sup>Department of Clinical Pathology and Oncology, <sup>2</sup>Department of Parasitology and Zoology and <sup>4</sup>Department and Clinic of Internal Medicine, Faculty of Veterinary Science, Szent István University, István utca 2, H-1078 Budapest, Hungary;  
<sup>3</sup>DuoBakt Veterinary Laboratory, Budapest, Hungary

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Pulmonary angiostrongylosis was diagnosed by the Baermann method and larval identification from faecal and bronchoalveolar lavage samples in a five-month-old male mongrel dog with dyspnoea and cough. Arterial blood gas analysis indicated arterial hypoxaemia and restrictive pneumopathy. In addition to the palliative treatment, fenbendazole was administered (50 mg/kg/24 h per os) for 14 days. The respiratory signs subsided within a short time clinically, but serial arterial blood gas analysis demonstrated an ongoing ventilation disorder. Repeated haematology, thoracic radiography, bronchoscopy and blood gas analysis were performed to follow the course of the disease. The most severe eosinophilia was detected after the beginning of the anthelmintic therapy, and the arterial pO<sub>2</sub> level was permanently low. Arterial blood gas analysis provided the most adequate information about the course of the pneumopathy and it greatly facilitated the patient's medical management.

**Key words:** Dog, bronchopneumonia, eosinophilia, pulmonary angiostrongylosis, arterial blood gas analysis, fenbendazole

Canine pulmonary angiostrongylosis (CPA) is caused by the nematode *Angiostrongylus vasorum*, which predominantly infects young canids (Kassai, 2003; Chapman et al., 2004; Nelson and Sellon, 2005). In Central Europe, the natural and reservoir host of *A. vasorum* is the red fox (*Vulpes vulpes*) (Kassai, 2003; Barutzki and Schaper, 2009). Although Hungary can be considered an endemic region, there have been few parasitological and clinical reports about CPA. Sréter et al. (2003) studied the prevalence of respiratory nematodes in cadavers of red foxes, and found *A. vasorum* in 5% of the cadavers examined. Majoros et al. (2010) conducted a parasitological survey of client-owned dogs in

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\*Corresponding author; E-mail: Csondes.Judit@aotk.szie.hu; Phone: 0036 (1) 478-4132; Fax: 0036 (1) 478-4269/8667

Baranya County and demonstrated *A. vasorum* infection in the faeces of two asymptomatic dogs by the Baermann method.

The intermediate hosts of *A. vasorum* are mostly slugs and snails (Nelson and Sellon, 2005), and the final hosts become infected by ingesting the intermediate hosts containing third-stage larvae (L3). The L3 get free in the small intestines and start their migration through the intestinal wall to the mesenteric lymph nodes. Here they develop to L4 and reach the liver with the lymphatic circulation or through the vena portae. In the liver they moult again to L5 and migrate to the right side of the heart or into the pulmonary arteries via the caudal vena cava. Adult worms are located mainly in the pulmonary arteries or in the right side of the heart (Kassai, 2003; Chapman et al., 2004; Koch and Willesen, 2009). The parasites reach their final habitat on postinfection days 9 or 10 and become sexually mature. In the pulmonary capillary network, their eggs turn into first-stage larvae (L1), which enter the alveoli and migrate toward the upper airways. The L1 are expectorated or passed through the gastrointestinal tract and discharged in the faeces. The prepatent period varies between one and two months but can be longer (Patteson et al., 1993; Garosi et al., 2005; Nelson and Sellon, 2005). CPA can be subclinical, accompanied by mild, chronic symptoms or it can be a life-threatening disease. In addition to general signs, the cardiorespiratory system, the blood coagulation and the central nervous system can all be affected (Patteson et al., 1993; Chapman et al., 2004; Garosi et al., 2005; Traversa and Guglielmini, 2008; Barutzki and Schaper, 2009). Larvae are generally identified in faecal or bronchoalveolar/transtracheal lavage samples by the Baermann method (Kassai, 2003; Chapman et al., 2004; Nelson and Sellon, 2005). Nowadays, an ELISA is also available for the detection of circulating *A. vasorum* antigens and/or specific antibodies in the blood (Schnyder et al., 2011; Schucan et al., 2012).

Angiostrongylosis can be treated with benzimidazoles, levamisole or macrocyclic lactones (Patteson et al., 1993; Chapman et al., 2004; Conboy, 2004; Nelson and Sellon, 2005). Massive destruction of the worms due to anthelmintic treatment (especially when using levamisole) may elicit an anaphylactic reaction (Koch and Willesen, 2009).

Arterial blood gas analysis contributes to determining the type and severity of different respiratory disorders and helps monitor the course of pneumopathies (Haskins, 2004; Ford and Mazzaferro, 2006). Arterial hypoxaemia is diagnosed if the arterial pO<sub>2</sub> is less than 90 mmHg (Haskins, 2004). Diffusion impairment develops due to the thickening of the alveolar-capillary membrane in diffuse pulmonary interstitial disease or due to the loss of alveolar or capillary surface area when pulmonary interstitial oedema, fibrosis, vasculitis or emphysema develops. Diffusion impairment is usually accompanied by low arterial pO<sub>2</sub> level and physiologic arterial pCO<sub>2</sub> level. These laboratory alterations reveal the restrictive nature of pulmonary disease (Haskins, 2004). When Beagle dogs were infected experimentally with *A. vasorum*, moderate hypoxaemia (median paO<sub>2</sub>

level of 73 mmHg and 74 mmHg, respectively) was detected 8 and 13 weeks after the inoculation (Kranjc et al., 2010).

The present study demonstrates the important role of serial arterial blood gas analysis in the clinical approach to a CPA case.

### Materials and methods

An approximately 5-month-old, non-vaccinated intact male mongrel dog was admitted to the Small Animal Clinic of the Faculty of Veterinary Science, Szent István University. The dog had been found 1.5 months before its hospitalisation in a rural part of North-eastern Hungary. Dyspnoea and coughing had started 1–2 weeks before, which deteriorated despite antibiotic and glucocorticoid treatment. Besides the physical examination, additional laboratory tests and instrumental examinations indicated a respiratory disorder.

For haematology and biochemistry, blood was obtained by venipuncture from the cephalic vein. For blood gas analysis (ABL 55 Blood Gas Analyser, Denmark) the femoral artery was punctured without anaesthesia or oxygen support to obtain an arterial blood sample according to Ford and Mazzaferro (2006). Arterial samples were analysed immediately. Abdominal ultrasonography, echocardiography and plain survey thoracic radiographs were also done. Urine was collected by cystocentesis and sent for PCR examination to exclude canine distemper infection. Whole blood was taken at the evening and the modified Knott's test was performed.

Bronchoscopic examination was carried out under general anaesthesia in sternal recumbency. A flexible videobronchoscope (diameter: 5.9 mm, length: 61 cm, working channel diameter: 2.3 mm; Karl Storz Germany) was used for the procedure. Bronchoalveolar lavage (BAL) was performed by wedging the bronchoscope into the smallest airways visible, followed by instillation and aspiration of 10 ml sterile saline infusion (Salsol inf., TEVA Pharmaceutical Works Co. Ltd., Hungary) through the working channel of the endoscope with a 20-ml sterile, single-use syringe (Romed Holland, Van Oostveen Medical B.V., The Netherlands). The collected sample was divided into EDTA-K3 tubes for cytology and parasitological examination and into a transport medium with charcoal (Transport Set, Biolab Inc., Hungary) for microbiology.

For cytology testing, the cell count of the BAL sample was measured using a haematology analyser (Abacus Junior Vet 5, Diatron MI PLC, Hungary). Subsequently the sample was added to BD Falcon, 15-mL Conical Centrifuge Tubes (Thermo Fisher Scientific Inc.) and then centrifuged (Universal 32 centrifuge, Hettich Instruments, LP) with 1,600 g for 5 min. Sample preparation was done as described by De Brauwer et al. (2000). The supernatant was discarded, and then the sediment was resuspended with the remaining fluid and pipetted

onto a glass slide. Sediment smears were prepared using a technique similar to that applied for blood smears. The samples were air dried at room temperature and stained with Accustain<sup>®</sup>, Giemsa stain (Sigma-Aldrich<sup>®</sup>). Microscopic examination was done using a binocular CX 21 Olympus microscope.

After receiving the BAL sample for microbiological evaluation, it was homogenised and spread onto Columbia agar with 5% sheep blood (V, bioMérieux, Marcy l'Etoile, France), Eosin Methylene Blue agar (EM, bioMérieux, Marcy l'Etoile, France) and Sabouraud-glucose agar with yeast extract (Sab, Bak-teszt Ltd., Hungary) according to the current guidelines (Czirók, 1999; Quinn et al., 2011). The plates were incubated at 37 °C for 24 h in normal oxygen atmosphere, and then left at room temperature for another 24 h; therefore, they were examined twice for bacterial growth. In the course of routine parasitological examination performed in the DuoBakt Laboratory, the faeces was suspended in 0.9% NaCl solution and samples were prepared by flotation technique (calcium nitrate solution of 1,270 g/cm<sup>3</sup> density; Molar Ltd., Hungary). The wet mount preparations were examined under × 100 and × 400 magnifications, while the smears prepared with surface enrichment were examined under × 60 and × 100 magnifications with an optical microscope. Because of the clinical suspicion, larval isolation was also performed using the Baermann method (Foreyt and Foreyt, 2002; Kassai, 2003; Zajac and Conboy, 2007).

Larval identification from faecal and BAL samples was performed at the Department of Parasitology and Zoology, Faculty of Veterinary Science, Szent István University. The faecal samples collected from the dog were divided into small parts and baermannised in conical glasses for 24 h. The native slimy bronchoalveolar mucous sample was spread between two glasses and visualised in a light microscope for parasitological investigation.

Hospital care included palliative support (intravenous fluid therapy, parenteral amoxicillin-clavulanic acid combination, aminophylline, bromhexine, vitamins) and anthelmintic treatment (fenbendazole in a dose of 50 mg/kg/24 h per os) for 14 days.

## Results

The poorly developed dog weighed 8.4 kg (body condition score: 2/5) and was in a bad general condition. Rectal body temperature was 39.0 °C, the respiratory rate was 100/min and the femoral pulse frequency was 150/min. A moderate amount of serous nasal discharge was continuously dripping from both nostrils. The buccal mucosa was slightly pale and the capillary refill time was 1.5 sec. The pharyngeal mucosa was reddened and covered with white foamy discharge. Petechiae or ecchymosis were not detected. The mandibular lymph nodes were slightly enlarged but the other regional lymph nodes had normal size. At rest the dog suffered from moderately severe mixed-type tachydyspnoea

which deteriorated during motion and excitement but cyanosis was absent. The recurrent productive cough was easy to provoke and it ended in retching. Above the trachea mild narrowing sounds could be heard during auscultation. Above the middle and the dorsal parts of the thoracic cavity intensified bronchial respiratory sounds and mild crackling sounds were auscultated symmetrically. Over the ventral third of the thorax weak alveolar respiratory sounds were detected. The heart rate was 150/min without arrhythmia or cardiac murmur. The abdominal cavity was slightly distended, but the undulation test was negative. During deep palpation of the abdomen sensitive intestinal loops were detected. The dog was weak but conscious. The locomotor and neurological systems were found intact by physical examination.

Haematological examination revealed normocytic normochromic anaemia and leukocytosis with marked eosinophilia. The platelet count was physiological (Table 1). Alkaline phosphatase activity was moderately elevated, while other biochemical parameters were within the reference interval. The clotting times (APTT, PTT) were physiological.

The orthogonal thoracic radiographs revealed a diffuse non-structured interstitial pulmonary infiltrate showing a patchy distribution over the whole lung area. A marked peribronchial infiltrate was also apparent around the large bronchi. The entire trachea had decreased diameter (the tracheal diameter/thoracic inlet ratio was 0.13). The cardiac silhouette was unremarkable (Fig. 1).

During abdominal ultrasonography enlarged mesenteric lymph nodes and thickened intestinal walls were detected. PCR examination of the urine for canine distemper virus infection was negative. On echocardiographic examination, the heart had a physiological shape and the cardiac chambers had normal diameters and wall thicknesses. The shape and movement of the interventricular septum, just like the diameter of the main pulmonary artery and the shape and movement of the pulmonary valves, were also normal. There was no detectable tricuspid or pulmonary regurgitation during the Doppler interrogation of these structures. Based on these findings, pulmonary hypertension was excluded. The modified Knott's test was negative.

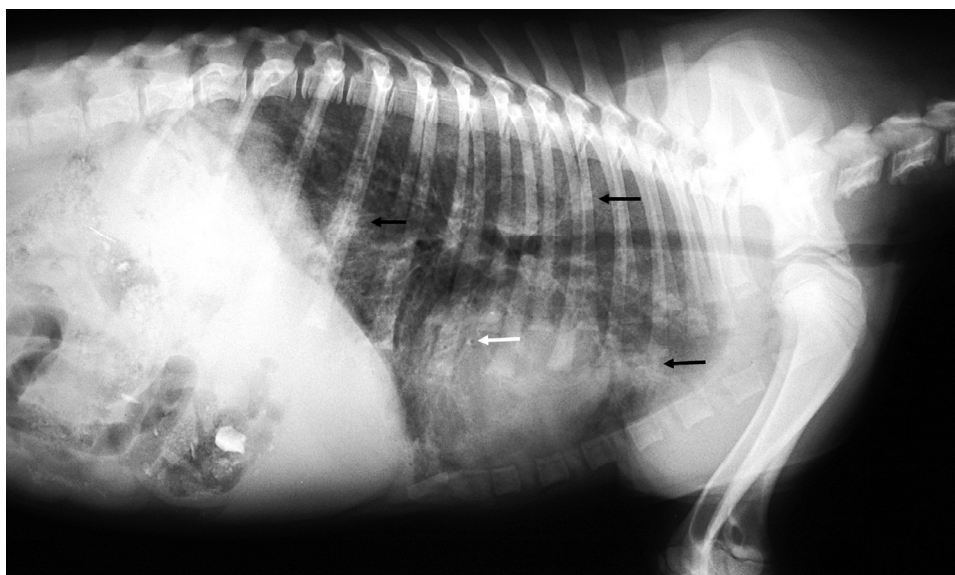
By bronchoscopy, a small amount of yellowish, sticky mucopurulent discharge was detected in the trachea and in the cavities of the major lobular bronchi (Fig. 2). Cytological evaluation of the bronchoalveolar mucus revealed that the specimen was rich in cells (Fig. 3). The total nucleated cell count was  $524 \times 10^9/l$ . Ciliated (25%) and non-ciliated (15%) columnar epithelial cells were found in the sample. Some ciliated (10%) and non-ciliated (7%) cuboidal epithelial cells and goblet cells (2–5%) were also visible. Alveolar macrophages were present in a ratio of 26–30%. Neutrophil granulocytes, generally segmented forms, appeared in 6–8%. Eosinophil granulocytes were also visible in moderate numbers

**Table 1**  
Haematology results

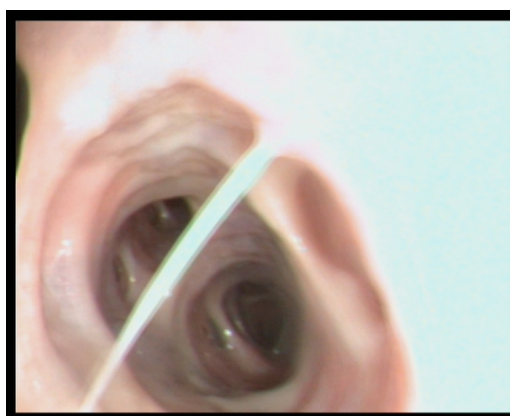
	Reference range*	12 October	First control (22 October) the 2nd day of fenbendazole therapy	Second control (3 November) the end of fenbendazole therapy and the beginning of prednisolone administration	Third control (15 December)
<b>Complete blood count</b>					
RBC	5.5–8.5 T/L	4.75 T/L	4.9 T/L	4.9 T/L	5.6 T/L
Haemoglobin	120–180 g/L	116 g/L	117 g/L	123 g/L	137 g/L
Haematocrit	0.35–0.55 L/L	0.33 L/L	0.34 L/L	0.34 L/L	0.37 L/L
MCV	60–80 fL	70.2 fL	68.5 fL	68 fL	67 fL
MCH	20–25 pg	24.3 pg	23.8 pg	25.1 pg	24.6 pg
MCHC	320–360 g/L	346 g/L	348 g/L	366 g/L	368 g/L
WBC	6.0–12.0 G/L	19.93 G/L	26.68 G/L	10.1 G/L	11.9 G/L
Neutrophils (stab form)	0–5%	2%	2%	–	2%
Absolute	0–0.3 G/L	0.93 G/L	0.53 G/L	–	0.24 G/L
Neutrophils (segment form)	60–80%	38%	35%	54%	54%
Absolute	3.0–11.0 G/L	7.57 G/L	9.34 G/L	5.45 G/L	6.43 G/L
Eosinophils	1–6%	26%	38%	10%	12%
Absolute	0.1–0.3 G/L	5.18 G/L	10.14 G/L	1.01 G/L	1.43 G/L
Lymphocytes	20–40%	31%	24%	34%	32%
Absolute	1.0–5.0 G/L	6.17 G/L	6.4 G/L	3.43 G/L	3.8 G/L
Monocytes	2–4%	3%	–	2%	–
Absolute	0.2–1.5 G/L	0.59 G/L	–	0.2 G/L	–
Platelet count	200–800 G/L	511 G/L	529 G/L	423 G/L	343 G/L
<i>Babesia</i>		–	–	–	–

\*T. Gaál (ed.): Veterinary Clinical Laboratory Diagnostics [in Hungarian]. Sík Kiadó, Budapest, 1999

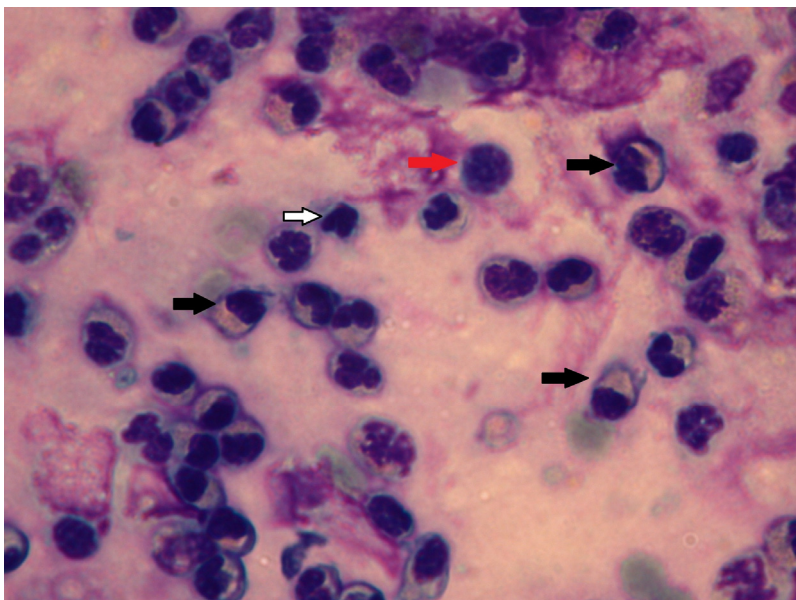
(10–12%). In one of the smears a lungworm larva (length 360.4  $\mu\text{m}$ , width varying between 12.9–17.9  $\mu\text{m}$  with a cephalic button at the apical end) was detected (Fig. 4). This was coiled from one end. It had a cephalic button at the apical end and the tail terminated in a sinus wave curve ('severe kink') with a wanly visible dorsal spine. Based on the cytological examination a subacute proliferative inflammation and lungworm infection were demonstrated. The microbiological examination of the BAL sample was negative.



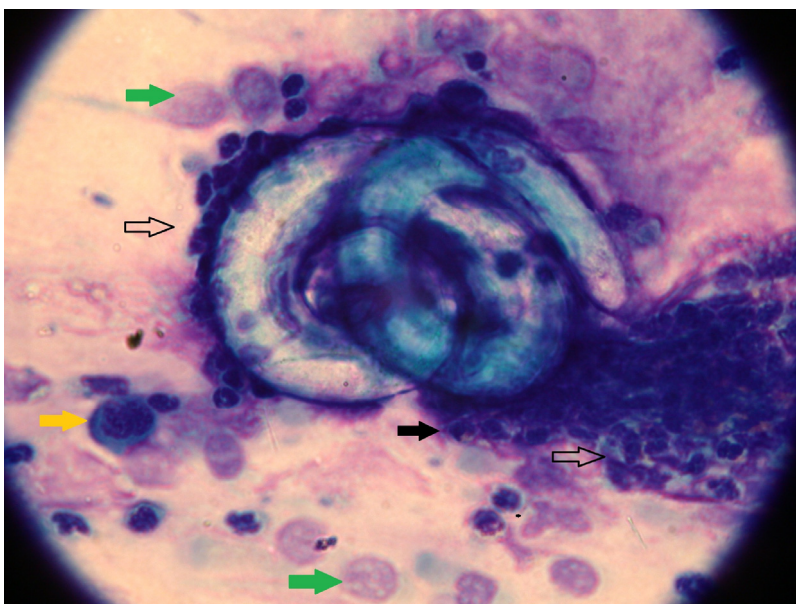
*Fig. 1.* The thoracic X-ray revealed patchy distribution of areas with diffuse interstitial pulmonary pattern (black arrows) and pronounced peribronchial infiltration around the large bronchi (white arrow)



*Fig. 2.* The appearance of bronchi during endoscopy. Note the mucosal irregularity and the slightly yellowish colour of the secretion



*Fig. 3.* Principally eosinophil (full black arrow) and neutrophil (black frame arrow) granulocytes are seen, with the occasional presence of small lymphocytes (red arrow), in the bronchoalveolar lavage sample by cytological evaluation



*Fig. 4.* Principally cuboidal epithelial cells (green arrow), alveolar macrophages (orange arrow), neutrophil (black frame arrow) and eosinophil granulocytes (full black arrow) and the lungworm larva are seen in the bronchoalveolar lavage sample



The parasitological investigation of BAL samples revealed some living and dead forms of larval nematodes; however, these free larvae and eggshells with contorted larvae within them were not suitable for larval identification. In the faecal sample only free and living larvae could be detected. The free larvae were slightly bigger than the unhatched ones, and they contained much more granules. Their length reached 260–360  $\mu\text{m}$  and the refractive granules accumulated mainly in the rear half of the body. The more or less clear oesophagus and the slightly darker intestine were of equal size, as their junction could be detected at the middle of the total length. The caudal third of the oesophagus was slightly swollen in front of the junction with the intestine. The head of larvae had a button-like protuberance on it. The larvae had a tapered end of tail which narrowed unequally, moving the tip of the tail slightly towards the dorsal side (Fig. 5). Only 12 specimens of parasitic nematode larvae were found on the bottom of the glasses at the end of the isolation period.



Fig. 5. *Angiostrongylus vasorum* larva detected by the Baermann technique

Arterial blood gas analysis revealed a mild ventilation disorder at the time of admission to the hospital. The arterial  $\text{pCO}_2$  ( $\text{paCO}_2$ ) value was within the reference range (39.9 mmHg), while the arterial  $\text{pO}_2$  ( $\text{paO}_2$ ) level was moderately low (75.6 mmHg). The  $\text{PaO}_2/\text{FiO}_2$  ratio was 360 (Table 2). Based on the results of analysis, restrictive pneumopathy with arterial hypoxaemia was diagnosed. On the second day of fenbendazole treatment, haematology and blood gas analysis were repeated. Eosinophilia was getting more severe and the arterial hypoxaemia was still present: the  $\text{paO}_2$  level was low (84.4 mmHg) and the  $\text{paCO}_2$  value remained within the physiologic range (38.6 mmHg). The  $\text{PaO}_2/\text{FiO}_2$  ratio was 401.9 (Table 2).

**Table 2**  
Results of the arterial blood gas analyses

	Reference range*	12 October	First control (22 October)	Second control (3 November)	Third control (15 December)
pH	7.35–7.45	7.39	7.41	7.39	7.39
pCO <sub>2</sub> (mmHg)	36–44	39.9	38.6	38.5	36.6
pO <sub>2</sub> (mmHg)	90–100	75.6	84.4	77.6	92.3
PaO <sub>2</sub> /FiO <sub>2</sub>	476	360	401.9	369.5	439.5
HCO <sub>3</sub> (mmol/l)	20–25	22.8	23.7	22.8	21.5
ABE (mmol/l)	±3.5	–0.7	0.3	–0.7	–1.6
K <sup>+</sup> (mmol/l)	3.5–5.5	4.2	4.2	4.2	4.1
Na <sup>+</sup> (mmol/l)	135–155	144	145	146	145
Ca <sup>2+</sup> (mmol/l)	0.8–1.5	1.02	1.12	1.17	1.25

\*Ford, R. B. and Mazzaferro, E. M. (2006): Kirk and Bistner's Handbook of Veterinary Procedures and Emergency Treatment. Elsevier, St. Louis, pp. 508–509

As a result of intensive care, the patient's condition markedly ameliorated. Tachydyspnoea and coughing improved, the respiratory sounds above the chest were still harsh but less than before, and the crackling noises ceased. Fenbendazole therapy (50 mg/kg/24 h per os altogether for 14 days) combined with palliative support (oral administration of aminophylline, multivitamin and probiotics) was continued at home.

On the 14th day of fenbendazole therapy the dog's general condition improved and the respiratory signs ameliorated. The faecal analysis (samples were collected on three consecutive days) was negative by the Baermann technique. On the orthogonal thoracic radiographs the lungs had the same pulmonary pattern but the general radiopacity of the thorax decreased (Fig. 6). Repeated echocardiography did not detect pulmonary hypertension. During repeated bronchoscopy and BAL sampling the mucosal surface of the lower respiratory tract was covered with a small amount of sticky discharge and the mucosa was slightly irregular, but amelioration was demonstrated as compared to the first endoscopic findings. Baermann isolation from the BAL sample was negative. By cytological evaluation the BAL sample was slightly cellular, with a slightly increased mucous production and normal epithelial production as signs of a chronic inflammatory process.

Repeated complete blood count revealed that the eosinophilia moderated (Table 1) and in the actual arterial blood gas sample arterial hypoxaemia still persisted. The paO<sub>2</sub> level was low (77.6 mmHg) and the paCO<sub>2</sub> value remained in the normal range (38.5 mmHg). The PaO<sub>2</sub>/FiO<sub>2</sub> ratio was 369.5 (Table 2).

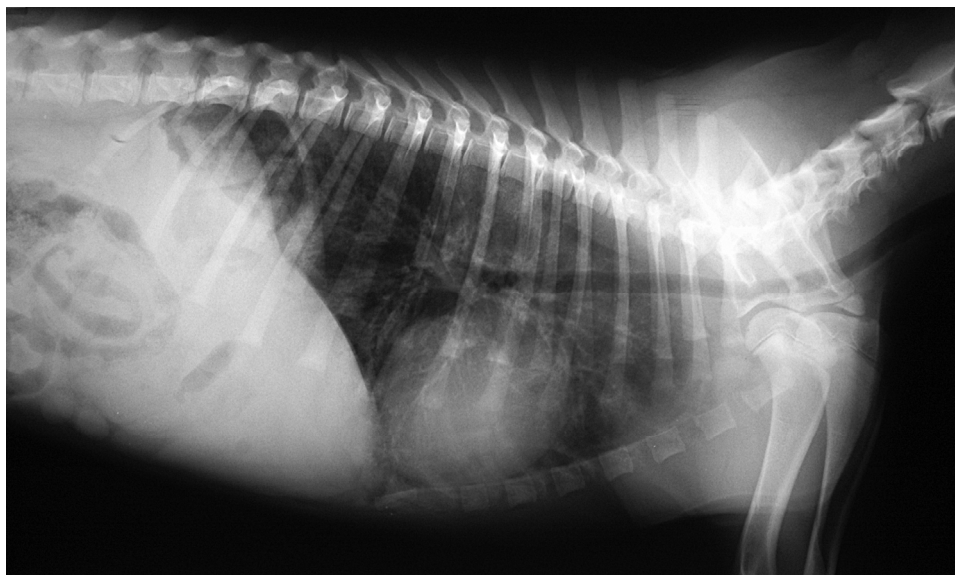


Fig. 6. The control thoracic radiograph. Note amelioration of the radiopacity as compared to the first X-ray picture

Fenbendazole administration was terminated. Prednisolone (0.5 mg/kg per os in the mornings) was started with H<sub>2</sub>-receptor blocker (famotidine, 0.5 mg/kg/12 h per os) to prevent the supposed ongoing inflammation-related fibrosis in the lungs. A local inflammatory process triggered by the dying/dead lungworms in the pulmonary tissue was suspected by this time, because the paO<sub>2</sub> level and PaO<sub>2</sub>/FiO<sub>2</sub> ratio were still decreased with elevated eosinophil numbers in the blood.

The final control examination was two months after the dog was released from the Intensive Care Unit. By that time the dog was asymptomatic, with no pathological respiratory sounds being detected over the thoracic cavity. Elevated numbers of eosinophil granulocytes were present in the blood (Table 1) but the level of paO<sub>2</sub> was physiological (92.3 mmHg) with an almost normal PaO<sub>2</sub>/FiO<sub>2</sub> ratio of 439.5 (Table 2). Bronchodilator therapy was discontinued and administration of prednisolone was gradually tapered off.

## Discussion

Bronchopneumonia and eosinophilia related to *A. vasorum* infection were diagnosed in our patient. Diseases which can potentially cause respiratory disorders and/or eosinophilia in a young, non-vaccinated dog were considered. There are many viral respiratory infections which can cause pneumonia (canine distemper virus; canine adenovirus type 2, canine respiratory coronavirus, canine

influenza virus, canine parainfluenza virus). Canine distemper was excluded by PCR from a urine sample because of its clinical importance. Bacterial infections (e.g. *Bordetella bronchiseptica*, *Klebsiella* spp., *Pasteurella multocida*, *Pseudomonas* spp., *Streptococcus* spp., *Staphylococcus* spp., *Mycoplasma* spp., *Chlamydia* spp.) of the respiratory tract or pulmonary aspergillosis were not confirmed by microbiological culture from the BAL sample. Heartworm disease was excluded by echocardiography and modified Knott's test. Faecal analysis did not reveal *Toxocara* or *Ancylostoma* infestation which can be accompanied by respiratory signs. An *A. vasorum* larva was revealed by larval isolation and distinguished from other respiratory parasites (e.g. above all *Crenosoma vulpis*, but also *Oslerus osleri*, *Filaroides* spp. and *Capillaria aerophila*). There are several non-infectious diseases which can cause respiratory disorders and/or eosinophilia (e.g. eosinophilic bronchopneumopathy, allergy, paraneoplastic disorder, primary ciliary dyskinesia, eosinophilic leukaemia, mastocytoma, panostitis, eosinophilic myositis, hypoadrenocorticism, lead toxicosis).

The effect of anthelmintic therapy was followed up using several additional laboratory methods and diagnostic imaging techniques: Baermann test, haematology, arterial blood gas analysis, echocardiography, bronchoscopy and thoracic radiographs. Repeated Baermann examination recommended by Koch and Willesen (2009) and Paradies et al. (2013) was carried out from a faecal sample on the 14th day of fenbendazole therapy and about 6 weeks after the end of fenbendazole treatment, and they did not reveal persistent *A. vasorum* infection. We found that fenbendazole was effective and safe for treating pulmonary angiostrongylosis.

In accordance with the echocardiographic results found in the experimental work of Kranjc et al. (2010), neither abnormalities in cardiac morphology and function nor pulmonary hypertension were detected in our patient.

Determination of the level of eosinophil granulocytes in the blood, thoracic radiographs and physical examination provided useful information about the course of the disease, but with serial blood gas analysis the inflammatory process and its effect on ventilation could be followed more precisely (Fig. 7). From the arterial blood gas levels the restrictive character of the pathological process was concluded, which is presumably caused by the affected pulmonary parenchyma (Haskins, 2004). The lowest  $\text{paO}_2$  level could be explained by the local inflammatory process triggered by the dying/dead lungworms in the pulmonary tissue. The ongoing pulmonary inflammation might have caused pulmonary fibrosis. To prevent this pathological process, glucocorticoid was administered in an anti-inflammatory dose. After 6 weeks of prednisolone administration the repeated arterial blood gas analysis showed a pronounced improvement (Table 2).

Serial arterial blood gas analysis was an adequate and sensitive method for following the course of restrictive pneumopathy associated with angiostrongylosis, and it substantially contributed to the patient's successful medical management.

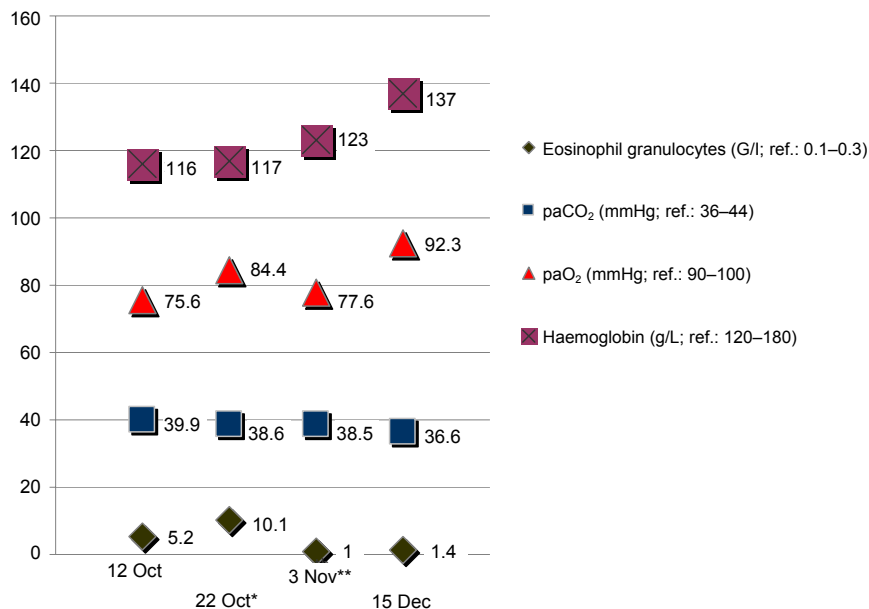


Fig. 7. Changes of several laboratory parameters during the course of pulmonary angiostrongylosis. Explanations: \*day 2 of fenbendazole therapy; \*\* the end of fenbendazole treatment and the beginning of prednisolone administration

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