



Case Report

A CASE OF HYDROTHORAX IN A DOG – CLINICAL, BLOOD LABORATORY AND ELECTROCARDIOGRAPHIC CHANGES

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ABSTRACT

The present report describes a clinical case of bilateral hydrothorax in a dog with chronic enteropathy. Significant deviations in blood biochemical parameters, the radiological and electrocardiographic findings in the studied punctate are reported. In our view, these alterations were important and could be successfully used in the diagnosis of this pathological state.

Key Words: pleural effusion, hydrothorax, dogs, haematology, electrocardiography

INTRODUCTION

The visceral pleura is a thin membrane encompassing the lung parenchyma. It plays a major role in the absorption of fluids, produced by the parietal sheet. The enhanced release of fluids and/or the reduced absorption capacity of the visceral pleura result in the accumulation of excess fluid in the pleural space (1, 2, 3).

According to the type of accumulated fluid, pleural effusions could be: accumulation of serous fluid (hydrothorax), blood (haemothorax), chyle (chylothorax) and pus (pyothorax) (4). The mechanisms involved in these events are capillary pressure, permeability of pleural capillaries, oncotic pressure and the lymphatic drainage of the thorax (1, 5, 6).

Pleural effusion is uncommon in carnivores (7). Although the cause of the pleural effusion may be readily apparent, such as when it is associated with cardiac disease, oftentimes the underlying disease is obscure and difficult to ascertain. Despite extensive diagnostic methods, in the majority of the pets, the main aetiology is undetermined – idiopathic effusions (8, 9). Maskell, N. A. (2003) reports that the aetiology of pleural effusions remains unknown in up to 15% of men (10).

Any breed dog or cat may be affected.

A breed predisposition has been suspected in the Afghan hound and Shiba Inu. Among cats, Oriental breeds such as Siamese and Himalayan are targets of increased prevalence.

CASE REPORT

A clinical case of hydrothorax in a dog - 8-year old female German Shepherd, weighing 30 kg, is described. The dog is owned by a private owner and was referred to the Small Animal Clinic of the Faculty of Veterinary Medicine at the Trakia University at December 27, 2007. According to the patient's history, the dog has been losing weight for several months, exhibited increased appetite and thirst, diarrhoeic stools and general weakness. Subsequently its owner noticed accelerated and difficult breathing with rapid emaciation and lack of appetite.

The physical examination of the patient revealed a marked respiratory distress. The respiration was labial and difficult in both phases, mainly of abdominal type. The dog was reluctant to move and became exhausted very rapidly. The clinical examination showed a rectal body temperature of 37.2°C; heart rate of 150 min⁻¹, weak and hardly perceptible, respiration rate of 45 min⁻¹; the lymph nodes appeared healthy. The visible mucous coats (conjunctival) were markedly cyanotic, and the capillary refill time was 4-5s. The elasticity of the skin was reduced, with marked enophthalm. The auscultation of the heart revealed dumb and indistinct cardiac

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tones. The lung auscultation showed lack of respiratory sounds in the lower half of the chest with enhanced vesicular breathing in the dorsal pulmonary areas. The abdomen was flat, non painful, with firm elastic consistence. The stools were extremely watery, with a dark colour, putrefactive odour and mucoid

plaques.

The radiography showed a dense shadow with horizontal upper margin in the ventral third of the thorax, an indistinct cardiac border and enhanced bronchial pattern (**Figure 1**)

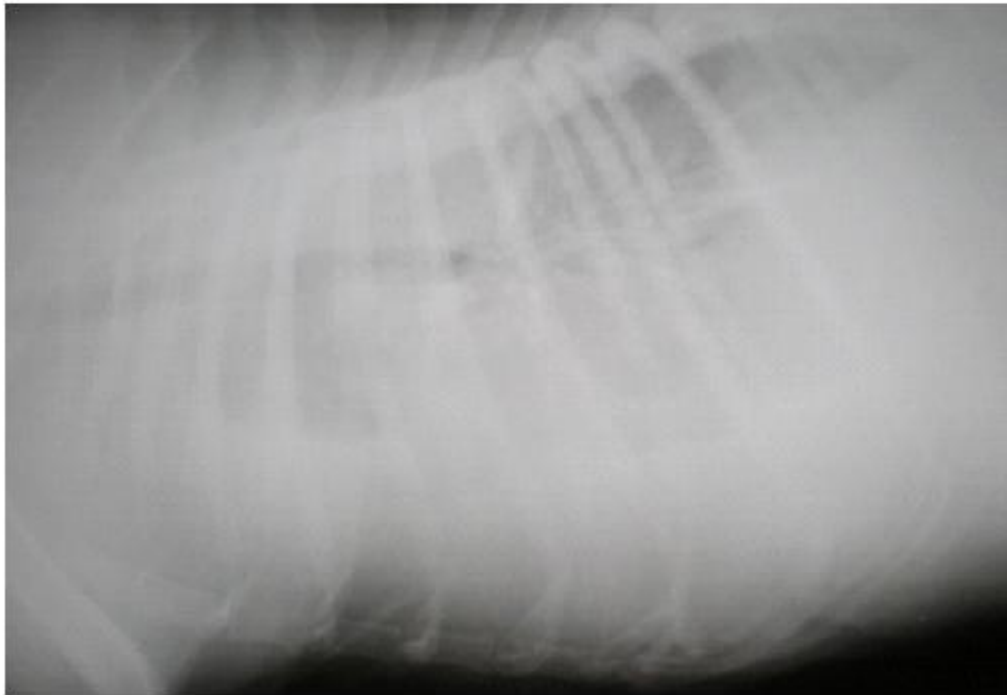


Figure 1. Thoracic effusion – increased ventral lung opacity with horizontal upper border.

The electrocardiography (ECG) showed a sinus tachycardia - HR 167 min^{-1} with obvious low-amplitude QRS complex, about 0.3 mV (**Figure 2**). The radiographic and ECG findings guided us to suspect a pleural effusion. The tentative diagnosis of pleural

effusion was confirmed by bilateral thoracocentesis and the large amount of aspirated punctate. The latter was determined as transudate by routine laboratory methods (**Table 1**). The bacteriological examination of punctate was negative.

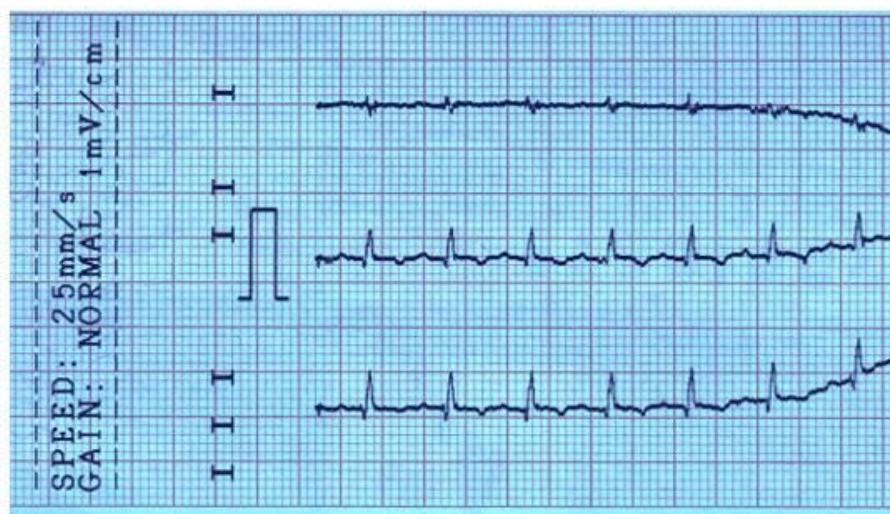


Figure 2. Sinus tachycardia with low-voltage QRS complex

Table 1. Data from the physical, chemical and cytological analyses of punctate.

Amount of punctate, ml/kg body weight	Colour	Transparency	Specific density	Protein content, g/L	Amount of blood cells
107	straw-yellow	transparent	1.013	17	-

The morphological blood analysis established erythrocytosis with hyperchromaemia and increased haematocrit values (**Table 2**). Leukocyte and thrombocyte counts were within the reference range. There was

neutrophilia (80%) with a left shift (**Table 3**). Blood biochemical analysis showed a considerable hypoproteinaemia (44 g/l) with hypoalbuminaemia and increased activities of liver transaminases (**Table 4**).

Table 2. Blood morphology

Haemoglobin, g/L	Erythrocytes T/L	Haematocrit, %	Leukocytes, G/L	Platelets, G/L
237	9.39	65.1	13.2	147

Table 3. Differential white blood cell counts

Eo %	Ba %	Mo %	Mm %	St %	Sg %	Ly %
3	1	4	3	5	72	12

Table 4. Blood biochemical parameters

Total protein, g/L	Albumin, g/L	ASAT U/L	ALAT U/L	Creatinine, μ mol/L	Urea, mmol/L	Total bilirubin, μ mol/L	Blood glucose, mmol/L
44	21	74	115	65.9	4.28	6.54	5.91

DISCUSSION

The commonest cases of hydrothorax are related to systemic protein deficiency (hypoproteinaemia), following glomerulonephropathy, renal amyloidosis, enteropathy or reduced liver synthesis of albumin (5, 7). Other frequent causes for transudate effusion in the pleural cavity are the right-sided heart failure with venous congestive events and thoracic neoplasm (11).

The extent of manifested clinical signs in hydrothorax correlates with the amount of fluid, the systemic compensatory capacity and the underlying disease that caused the effusion (12, 13, 14). The extensive amount of transudate in the thorax (**Table 1**) results in pulmonary compression and collapse and consequently, to signs of severe respiratory failure. In an insignificant pleural effusion (under 20 ml fluid/kg b.w.) there are no apparent respiratory troubles. The moderate effusion (20-40 ml/kg b.w.) is accompanied by dyspnoea on physical exertion. The massive effusion (over 100 ml/kg b.w.) is accompanied by tachypnoea, shallow breathing, dyspnoea, barrel-chest and orthopnoic body position. Also, pale or cyanotic mucous coats, fading or absent heart tones and respiratory sounds, dullness with horizontal upper border in percussion could be observed (5).

The registered sinus tachycardia is a compensatory mechanism of accelerated and difficult breathing, resulting in sympathetic nervous system excitation. The mentioned

causes exert their effect by a direct influence on the sinus node (15). The low voltage of the R peak of 0.3 mV (**Figure 2**) is a consequence of the relative higher distance between the heart and the electrodes (16). Similar events could be seen in effusions (pericardial, pleural or ascitis), hypothyreosis, hypokalaemia, pneumothorax and hypovolaemias (17).

The blood picture revealed a significant erythrocytosis, hyperchromaemia and increased haematocrit. These changes were mostly due to the occurring dehydration following the accumulation of a large amount of transudate in the thorax. To a certain extent, the changes in these parameters could be attributed to compensatory mechanisms in the attempts of the organism to maintain an optimal gas exchange in tissues and cells. The lack of significant changes in leukocyte counts (13 G/l) indicated the lack of inflammation. The hypoproteinaemia and hypoalbuminaemia are a result of impaired absorption function of intestinal epithelium and the developed chronic enteropathy (18). Due to the impaired digestion in the intestinal lumen and the formation of a number of toxic substances, a damage of liver parenchyma occurred that resulted in higher activities of transaminases (ASAT and ALAT). The lack of changes in blood urea and creatinine concentrations as well as in urinalysis (**Table 5**), allowed us to reject the hypothesis of nephropathy as a possible cause for hypoproteinaemia.

Table 5. Urinalysis in a dog with hydrothorax

Specific density	Nitrite	pH	Protein	Glucose	Ketones	Urobilinogen	Bilirubin	Leuc	Er	Hb
1.030	Neg	6	Neg	Normal	Neg	Normal	Neg	Neg	+	Neg

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